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OM protein - protein search, using sw model

February 28, 2004, 07:03:55; Search time 71.5 Seconds Run on:

(without alignments)

31.614 Million cell updates/sec

US-09-668-314C-84 Title:

Perfect score: 41

1 LVFFAEDF 8 Sequence:

Scoring table: BLOSUM62

Gapop 10.0 , Gapext 0.5

1586107 seqs, 282547505 residues Searched:

1586107 Total number of hits satisfying chosen parameters:

Minimum DB seq length: 0

Maximum DB seq length: 2000000000

Post-processing: Minimum Match 0%

Maximum Match 100%

Listing first 1000 summaries

A Geneseq_29Jan04:* Database :

1: geneseqp1980s:*

2: geneseqp1990s:*

3: geneseqp2000s:*

4: geneseqp2001s:*

5: geneseqp2002s:* 6: geneseqp2003as:*

7: geneseqp2003bs:*

8: geneseqp2004s:*

Pred. No. is the number of results predicted by chance to have a score greater than or equal to the score of the result being printed, and is derived by analysis of the total score distribution.

SUMMARIES

		ક				
Result		Query				
No.	Score	Match	Length 1	DB	ID	Description
						Aae10662 Human amy
1	41	100.0	8	4	AAE10662	
2	41	100.0	8	4	AAE02614	Aae02614 Human amy
3	35	85.4	8	2	AAR08190	Aar08190 Cerebrova
4	35	85.4	8	2	AAW32551	Aaw32551 Amyloidog
5	35	85.4	8	4	AAE10663	Aae10663 Human amy
6	35	85.4	8	4	AAE02615	Aae02615 Human amy
7	35	85.4	8	5	ABB78624	Abb78624 Human alp
8	35	85.4	8	5	ABB78623	Abb78623 Human alp
9	35	85.4	8	6	ABU09765	Abu09765 Amyloidog

10	35	85.4	8	6	ABR61959	Abr61959 Human amy
11		85.4	8	7	ABW00134	Abw00134 Beta-amyl
12		85.4	9	6	ABU79063	Abu79063 Aggregati
13		85.4	9	7	ABW00197	Abw00197 Peptide #
		85.4		3	AAY79938	Aay79938 Beta-amyl
14			10		AAB46229	Aab46229 Human APP
15		85.4	10	4		Aab46226 Human APP
16		85.4	10	4	AAB46226	Aab46228 Human APP
17	35	85.4	10	4	AAB46228	Aab46227 Human APP
18	35	85.4	10	4	AAB46227	
19	35	85.4	11	2	AAW32560	Aaw32560 Anti-amyl
20	35	85.4	11	4	AAM52586	Aam52586 Peptide #
21	35	85.4	11	5	AAU99431	Aau99431 Human amy
22	35	85.4	11	5	AAE29504	Aae29504 Amyloid b
23	35	85.4	11	6	ABU79013	Abu79013 Amyloidog
24	35	85.4	11	7	ABW00147	Abw00147 Amyloid-b
25	35	85.4	12	2	AAR60372	Aar60372 Beta-amyl
26	35	85.4	12	3	AAB10957	Aab10957 Bovine AD
27	35	85.4	12	6	AAE35466	Aae35466 Abeta pep
28	35	85.4	13	6	AAE35465	Aae35465 Abeta pep
29	35	85.4	13	6	AAE35467	Aae35467 Abeta pep
30	35	85.4	13	6	ADA37467	Ada37467 Human amy
31	35	85.4	14	4	AAE03423	Aae03423 Peptide c
32	35	85.4	14	6	ADA89887	Ada89887 Beta-A4 s
33	35	85.4	15	2	AAW02334	Aaw02334 Beta-amyl
34	35	85.4	15	2	AAW89358	Aaw89358 Beta-amyl
35	35	85.4	15	2	AAW89354	Aaw89354 Beta-amyl
36	35	85.4	15	5	ABG71014	Abg71014 Long form
37	35	85.4	15	5	ABB05162	Abb05162 Beta amyl
38	35	85.4	15	5	AAE26271	Aae26271 Human bet
39	35	85.4	15	6	ABU79057	Abu79057 Aggregati
40	35	85.4	15	6	ABU79064	Abu79064 Aggregati
41	35	85.4	15	6	ABU79058	Abu79058 Aggregati
42	35	85.4	15	6	ABU79055	Abu79055 Aggregati
43	35	85.4	15	6	ABU79056	Abu79056 Aggregati
44	35	85.4	15	6	ABU79062	Abu79062 Aggregati
45	35	85.4	15	7	ABW00192	Abw00192 Peptide #
46	35	85.4	15	7	ABW00190	Abw00190 Peptide #
47	35	85.4	15	7	ABW00198	Abw00198 Peptide #
48	35	85.4	15	7	ABW00189	Abw00189 Peptide #
49	35	85.4	15	7	ABW00191	Abw00191 Peptide #
50	35	85.4	15	7	ABW00196	Abw00196 Peptide #
51	35	85.4	16	5	AAE26330	Aae26330 Human bet
52	35	85.4	17	2	AAR54703	Aar54703 Beta-amyl
53	35	85.4	17	2	AAW18880	Aaw18880 Beta-amyl
	35	85.4	17	4	AAB91774	Aab91774 Amyloid b
54					AAB91807	Aab91807 Amyloid b
55	35	85.4	17	4		Aab48346 Beta-amyl
56	35	85.4	17	4	AAB48346	Abb04911 Human amy
57	35	85.4	17	5	ABB04911	Abb99611 Peptide d
58	35	85.4	17	6	ABB99611	Aab10963 Beta-amyl
59	35	85.4	18	3	AAB10963	
60	35	85.4	19	2	AAW18882	Aaw18882 AEDANS-be
61	35	85.4	19	2	AAW18881	Aaw18881 Trp-Beta-
62	35	85.4	19	3	AAY79935	Aay79935 Beta-amyl
63	35	85.4	19	4	AAB49097	Aab49097 Human amy
64	35	85.4	19	4	AAB46201	Aab46201 Human APP
65	35	85.4	20	3	AAY79934	Aay79934 Beta-amyl
66	35	85.4	21	2	AAY30941	Aay30941 Human sec

				_	77750500	n - u C O E C O N l ahoimor
67	35	85.4	24	2	AAR52569	Aar52569 Alzheimer
68	35	85.4	24	4	AAB91832	Aab91832 Amyloid b
69	35	85.4	24	4	AAB91805	Aab91805 Amyloid b
70	35	85.4	26	2	AAW47229	Aaw47229 Beta-amyl
71	35	85.4	26	2	AAY33408	Aay33408 Human amy
72	35	85.4	26	4	AAB84431	Aab84431 Partial s
73	35	85.4	26	6	ABU63718	Abu63718 Rat amylo
74	35	85.4	27	2	AAY33409	Aay33409 Human amy
75	35	85.4	28	1	AAP70594	Aap70594 Sequence
76	35	85.4	28	1	AAP90381	Aap90381 Synthetic
77	35	85.4	28	2	AAR60368	Aar60368 Beta-amyl
78	35	85.4	28	2	AAR54702	Aar54702 Beta-amyl
79	35	85.4	28	2	AAR64171	Aar64171 A4-P(1-28
80	35	85.4	28	2	AAR64164	Aar64164 Generic b
81	35	85.4	28	2	AAR64172	Aar64172 A4-B(1-28
82	35	85.4	28	2	AAR64170	Aar64170 A4-O(1-28
83	35	85.4	28	2	AAW01413	Aaw01413 Beta/A4-a
84	35	85.4	28	2	AAY39805	Aay39805 Beta-amyl
85	35	85.4	28	2	AAW81467	Aaw81467 Synthetic
86	35	85.4	28	4	AAB35591	Aab35591 Human clo
87	35	85.4	28	4	AAB35595	Aab35595 Human clo
88	35	85.4	28	4	AAB35594	Aab35594 Human clo
89	35	85.4	28	4	AAB35592	Aab35592 Human clo
90	35	85.4	28	4	AAB35593	Aab35593 Human clo
91	35	85.4	28	4	AAB35597	Aab35597 Human clo
92	35	85.4	28	4	AAB35596	Aab35596 Human clo
93	35 35	85.4	28	4	AAB35598	Aab35598 Human clo
			28	4	AAB35599 AAB35599	Aab35599 Human clo
94	35 35	85.4	28	4	AAB36202	Aab33333 Human clo
95	35	85.4				Aab35292 Human clo
96	35	85.4	28	4	AAB35590	Aab33330 Hullah Cis Aab91816 Amyloid b
97	35	85.4	28	4	AAB91816	Aab91789 Amyloid b
98	35	85.4	28	4	AAB91789	Aab91827 Amyloid b
99	35	85.4	28	4	AAB91827	Aab91783 Amyloid b
100	35	85.4	28	4	AAB91783	Aab91703 Amyloid b
101	35	85.4	28	4	AAB91800	Aab91800 Amy101d b Aab49396 Human amy
102	35	85.4	28	4	AAB49396	Aab49390 Human bet
103	35	85.4	28	5	AAE21439	Abb76030 Beta amyl
104	35	85.4	28	5	ABB76030	Abb76030 Beta amyr Aao18476 Human bet
105	35	85.4	28	5	AAO18476	
106	35	85.4	28			Aau76484 Amino aci
107	35	85.4	28	5	ABB04910	Abb04910 Human amy
108	35	85.4	28	5	AAE26081	Aae26081 Beta amyl
109	35	85.4	28	5	AAM50910	Aam50910 Beta amyl
110	35	85.4	28	5	ABB77991	Abb77991 Fragment
111	35	85.4	28	6	AAE35672	Aae35672 Human bet
112	35	85.4	28	6	AAE33794	Aae33794 Beta-amyl
113	35	85.4	28	6	ABG72238	Abg72238 Mutant H6
114	35	85.4	28	6	ABG72246	Abg72246 Mutant K2
115	35	85.4	28	6	ABG72234	Abg72234 Wild-type
116	35	85.4	28	6	ABG72235	Abg72235 Mutant D1
117	35	85.4	28	6	ABG72241	Abg72241 Mutant H1
118	35	85.4	28	6	ABG72240	Abg72240 Mutant El
119	35	85.4	28	6	ABG72237	Abg72237 Mutant R5
120	35	85.4	28	6	ABG72243	Abg72243 Mutant K1
121	35	85.4	28	6	ABG72242	Abg72242 Mutant H1
122	35	85.4	28	6	ABG72236	Abg72236 Mutant E3
123	35	85.4	28	6	ABG72239	Abg72239 Mutant D7

								_,
	124	35	85.4	28	6	AAE35431		Abeta pep
	125	35	85.4	28	6	AAE33219		Beta amyl
	126	35	85.4	28	6	ABU63712	Abu63712	Rat amylo
	127	35	85.4	28	7	AAE38831	Aae38831	Membrane
	128	35	85.4	29	5	AAE26331	Aae26331	Human bet
	129	35	85.4	30	2	AAW81468		Synthetic
	130	35	85.4	30	5	ABG94392		A beta pe
	131	35	85.4	30	5	AAU11766		Human amy
				30	5	ABG80717		Mouse Res
	132	35	85.4					Modified
	133	35	85.4	30	5	ABG80704	_	
•	134	35	85.4	30	6	ABR42769		Human amy
	135	35	85.4	32	4	AAB84430		Partial s
	136	35	85.4	33	2	AAW81469		Synthetic
	137	35	85.4	33	5	AAU93990		Human bet
	138	35	85.4	33	7	ADE10851		Chimeric
	139	35	85.4	35	2	AAW02336	Aaw02336	Beta-amyl
	140	35	85.4	35	2	AAW47228	Aaw47228	Beta-amyl
	141	35	85.4	35	2	AAW89361	Aaw89361	Beta-amyl
	142	35	85.4	35	2	AAW89357		Beta-amyl
	143	35	85.4	35	2	AAW89356		Beta-amyl
	144	35	85.4	35	2	AAW89359		Beta-amyl
			85.4	35	5	ABG71016		Long form
	145	35				ABB05164	_	EEVVHHHHQ
	146	35	85.4	35	5			Abeta pep
	147	35	85.4	35	6	AAE35430		
	148	35	85.4	36	2	AAW81471		Synthetic
	149	35	85.4	36	5	AAU11776		Synthetic
	150	35	85.4	36	5	AAU11771		Synthetic
	151	35	85.4	36	6	ABR42779		Amyloid b
	152	35	85.4	36	6	ABR42774		Amyloid b
	153	35	85.4	38	2	AAR60362	Aar60362	Beta-amyl
	154	35	85.4	38	2	AAW92722	Aaw92722	Human tac
	155	35	85.4	38	4	AAB91826	Aab91826	Amyloid b
	156	35	85.4	38	4	AAB91799	Aab91799	Amyloid b
	157	35	85.4	39	2	AAR60363		Beta-amyl
	158	35	85.4	39	2	AAW81472		Synthetic
	159	35	85.4	39	2	AAY25134		Human amy
	160	35	85.4	39	3	AAY52132		Human Rec
			85.4	39	6	ABU08509		Human amy
	161	35				ABP96148		Human Abe
	162	35	85.4	39	6		-	
	163	35	85.4	40				Beta-amyl
	164	35	85.4	40	2	AAR60364		Beta-amyl
	165	35	85.4	40	2	ADD11651		Human bet
	166	35	85.4	40	2	AAW23335		Amyloid b
	167	35 ,	85.4	40	2	AAW37507		Amyloid b
	168	35	85.4	40	2	AAW47226		Beta-amyl
	169	35	85.4	40	2	AAY14099	Aay14099	Human bet
	170	35	85.4	40	2	AAY39804		Beta-amyl
	171	35	85.4	40	2	AAW99584	Aaw99584	Wild type
	172	35	85.4	40	2	AAW81473		Synthetic
,	173	35	85.4	40	2	AAY39339		Beta-amyl
	174	35	85.4	40	2	AAY25135	-	Human amy
	175	35	85.4	40	2	AAW92723		Human tac
								Partial s
	176	35	85.4	40	4	AAB84426		Partial s
	177	35	85.4	40	4	AAB84429		
	178	35	85.4	40	4	AAB91786		Amyloid b
	179	35	85.4	40	4	AAB91813		Amyloid b
	180	35	85.4	40	4	AAB91819	Aab91819	Amyloid b

181	35	85.4	40	4	AAB91780	Aab91780 Amyloid b
182	35	85.4	40	4	AAB91792	Aab91792 Amyloid b
183	35	85.4	40	4	AAB91829	Aab91829 Amyloid b
						. Aab91802 Amyloid b
184	35	85.4	40	4	AAB91802	
185	35	85.4	40	4	AAE05483	Aae05483 Human pep
186	35	85.4	40	5	AAU99425	Aau99425 Human amy
187	35	85.4	40	5	AAE22990	Aae22990 Human amy
188	35	85.4	40	5	AAU11773	Aau11773 Synthetic
189	35	85.4	40	5	AAU11772	Aau11772 Synthetic
		85.4	40	5	AAG68313	Aag68313 Human bet
190	35					Aau96895 Human sel
191	35	85.4	40	5	AAU96895	
192	35	85.4	40	5	AAM50909	Aam50909 Beta amyl
193	35	85.4	40	5	AAU80186	Aau80186 Amyloid b
194	35	85.4	40	5	AAE26332	Aae26332 Human bet
195	35	85.4	40	5	AAM51863	Aam51863 Human amy
196	35	85.4	40	6	ABU08710	Abu08710 Amlyoid b
197	35	85.4	40	6	ABU08508	Abu08508 Human amy
198	35	85.4	40	6	AAO19885	Aao19885 Human amy
						Abp96147 Human Abe
199	35	85.4	40	6	ABP96147	
200	35	85.4	40	6	AAE35429	Aae35429 Abeta pro
201	35	85.4	40	6	ABP60626	Abp60626 Human A-b
202	35	85.4	40	6	ABP97883	Abp97883 Amino aci
203	35	85.4	40	6	ABR42775	Abr42775 Amyloid b
204	35	85.4	40	6	ABR42776	Abr42776 Amyloid b
205	35	85.4	40	6	ABU63706	Abu63706 Rat amylo
206	35	85.4	40	7	ADA37266	Ada37266 Human bet
207	35	85.4	40	7	ADB85563	Adb85563 Beta-amyl
208	35	85.4	40	7	AAE38648	Aae38648 Human amy
209	35	85.4		7	ADC66001	Adc66001 Human A(b
		85.4	40	7	ADC35182	Adc35182 Beta-amyl
210	35 35					Aar45230 Beta amyl
211	35	85.4	41	2	AAR45230	
212	35	85.4	41	2	AAR60365	Aar60365 Beta-amyl
213	35	85.4	41	2	AAR65283	Aar65283 Beta amyl
214	35	85.4	41	2	AAY25136	Aay25136 Human amy
215	35	85.4	41	3	AAB11497	Aab11497 Human amy
216	35	85.4	41	6	ABU08507	Abu08507 Human amy
217	35	85.4	41	6	ABP96146	Abp96146 Human Abe
218	35	85.4	42	1	AAP83153	Aap83153 Lambda SM
219	35	85.4	42	2	AAR10025	Aar10025 Beta-amyl
220	35	85.4		2	AAR20330	Aar20330 Sequence
221	35	85.4	42	2	AAR37867	Aar37867 Beta-amyl
222	35	85.4	42	2	AAR33192	Aar33192 Beta-amyl
223	35	85.4	42	2	AAR60366	Aar60366 Beta-amyl
		85.4	42	2	AAR65287	Aar65287 Beta amyl
224	35					Aar65288 Beta amyl
225	35	85.4	42	2	AAR65288	
226	35	85.4	42	2	AAR65285	Aar65285 Beta amyl
227	35	85.4	42	2	AAR65286	Aar65286 Beta amyl
228	35	85.4	42	2	AAR65284	Aar65284 Beta amyl
229	35	85.4	42	2	AAR95248	Aar95248 Beta/A4-a
230	35	85.4	42	2	AAR88206	Aar88206 Rat A42 b
231	35	85.4	42	2	AAR94591	Aar94591 Alzheimer
232	35	85.4	42	2	AAR99536	Aar99536 Murine be
233	35	85.4	42	2	AAW12828	Aaw12828 Beta A4 p
234	35	85.4	42	2	AAW64507	Aaw64507 Neurotoxi
235	35	85.4	42	2	AAW42989	Aaw42989 Full leng
236	35	85.4	42	2	AAW47230	Aaw47230 Beta-amyl
		85.4	42	2	AAW47230 AAY49691	Aay49691 Human bet
237	35	03.4	4 2	۷	MAINJUJI	ray 15051 Haman Dec

	238	35	85.4	42	2	AAW99585	Aaw99585	Mutant ag
	239	35	85.4	42	2	AAW81474		Synthetic
	240	35	85.4	42	2	AAY08607		Human bet
	241	35	85.4	42	2	AAW29093	-	A-beta-bi
	242	35	85.4	42	2	AAY25137		Human amy
	242	35	85.4	42	2	AAW92726	-	Human tac
			85.4	42	2	AAY33407		Human amy
	244	35		42	3		-	Beta-amyl
	245	35	85.4		3 4	AAY96956		Human Alz
	246	35	85.4	42		AAB86134		Beta/A4-a
	247	35	85.4	42	4	AAB35589		
	248	35	85.4	42	4	AAB49098		Human amy
	249	35	85.4	42	4	AAB84427		Partial s
	250	35	85.4	42	4	AAB48497		Human amy
	251	35	85.4	42	4	AAB91785		Amyloid b
	252	35	85.4	42	4	AAB91818		Amyloid b
	253	35	85.4	42	4	AAB91779		Amyloid b
	254	35	85.4	42	4	AAB91812		Amyloid b
	255	35	85.4	42	4	AAB91791		Amyloid b
	256	35	85.4	42	4	AAB82622	Aab82622	Amyloid-b
	257	35	85.4	42	4	AAB49395	Aab49395	Human amy
	258	35	85.4	42	4	AAB48830	Aab48830	Human amy
	259	35	85.4	42	4	AAE03425	Aae03425	Mouse amy
	260	35	85.4	42	4	AAE05484	Aae05484	Human pep
-	261	35	85.4	42	5	ABB81321		Amyloid p
	262	35	85.4	42	5	AAU80961		Human amy
	263	35	85.4	42	5	AAU98727		Human amy
	264	35	85.4	42	5	ABG94281		Amyloid b
	265	35	85.4	42	5	AAE21438		Human bet
	266	35	85.4	42	5	ABB76029		Beta amyl
	267	35	85.4	42	5	AAE25335		Modified
				42	5	AA015848		Beta-amyl
	268	35	85.4	42	5			Amino aci
	269	35	85.4			AAU76483		Beta amyl
	270	35	85.4	42	5	AAE26080		Human bet
	271	35	85.4	42	5	AAG68314		
	272	35	85.4	42	5	AAU96896		Human Amy
	273	35	85.4	42	5	AAU93988		Human bet
	274	35	85.4	42	5	AAE26300		Human bet
	275	35	85.4	42	5	ABG80593		Human amy
	276	35	85.4	42		AAM51864		Neuronal
	277	35	85.4	42				Amyloid p
	278	35	85.4	42	5	ABB83306		Amyloid-b
	279	35	85.4	42	5	ABB77990		Beta-amyl
	280	35	85.4	42	6	AAE35671		Human bet
	281	35	85.4	42	6	ABU08711		Amlyoid b
	282	35	85.4	42	6	AAO16344	Aao16344	A-beta pr
	283	35	85.4	42	6	ABU08506	Abu08506	Human amy
	284	35	85.4	42	6	AAE33793	Aae33793	Beta-amyl
	285	35	85.4	42	6	ABP99423	Abp99423	Beta-amyl
	286	35	85.4	42	6	ABB82633		Abeta fib
	287	35	85.4	42	6	ABP96144	Abp96144	Human Abe
	288	35	85.4	42	6	ABG72233		Human bet
	289	35	85.4	42	6	AAE35428		Abeta pro
	290	35	85.4	42	6	AAE33218		Beta amyl
		35 35	85.4	42	6	ABP97882		Amino aci
	291						_	Rat amylo
	292	35 25	85.4	42	6	ABU63707		Beta-amyl
	293	35	85.4	42	6	ADA74126		
	294	35	85.4	42	6	ADA89912	Ada89912	Abeta42 a

0.0.5	25	05.4	4.0	_	* D D O O C O	7h~02059	VEGF bind
295	35	85.4	42	6	ABR82058		
296	35	85.4	42	7	ADA37267		Human bet
297	35	85.4	42	7	ADB37652		Human bet
298	35	85.4	42	7	ADB85562		Beta-amyl
299	35	85.4	42	7	ADB75176	Adb75176	Amyloid b
300	35	85.4	42	7	AAE38649	Aae38649	Human amy
301	35	85.4	42	7	ADC66002		Human A(b
302	35	85.4	42	7	ADC35181		Beta-amyl
		85.4	42	7	ADD20743		Human bet
303	35						Chimeric
304	35	85.4	42	7	ADE10848		
305	35	85 .4	43	1	AAP96371		Region of
306	35	85.4	43	2	AAR54759		Beta amyl
307	35	85.4	43	2	AAR60367		Beta-amyl
308	35	85.4	43	2	AAR61328	Aar61328	Amyloid b
309	35	85.4	43	2	AAR64165	Aar64165	Beta amyl
310	35	85.4	43	2	ADD11650	Add11650	Human bet
311	35	85.4	43	2	AAR95673	Aar95673	A-beta pr
312	35	85.4	43	2	AAW93371		Human bet
		85.4	43	2	AAY17758		Beta-amyl
313	35					-	Natural b
314	35	85.4	43	2	AAW51316		
315	35	85.4	43	2	AAY42955		Beta-amyl
316	35	85.4	43	2	AAB21216		Beta-amyl
317	35	85.4	43	2	AAW71378		Beta-amyl
318	35	85.4	43	2	AAW40129	Aaw40129	Human amy
319	35	85.4	43	2	AAW92724	Aaw92724	Human tac
320	35	85.4	43	2	AAW89362	Aaw89362	Beta-amyl
321	35	85.4	43	3	AAY88390	Aay88390	Beta-amyl
322	35	85.4	43	3	AAY56102	-	Natural b
323	35	85.4	43	3	AAB27020	-	Beta-amyl
				3	AAB15372		Human bet
324	35	85.4	43				Beta-amyl
325	35	85.4	43	4	ABB07901		
326	35	85.4	43	4	AAB84428		Partial s
327	35	85.4	43	4	AAB91811		Amyloid b
328	35	85.4	43	4	AAB91778		Amyloid b
329	35	85.4	43	4	AAG78791		Human bet
330	35	85.4	43	4	AAB48344	, Aab48344	Beta-amyl
331	35	85.4	43	4	AAB81193	Aab81193	Beta-amyl
332	35	85.4	43	4	AAB98986	Aab98986	Beta-amyl
333	35	85.4	43	4	AAB47108	Aab47108	Biotinyla
334	35	85.4	43	4	AAE12508		Beta-amyl
335	35	85.4	43	5	ABB98516	Abb98516	Human bet
336	35	85.4	43	5	ABG71001		Natural 1
							Human bet
337	35	85.4	43	5	AAO18457		Beta amyl
338	35	85.4	43	5	ABB05149		_
339	35	85.4	43	5	AAU98701		Human amy
340	35	85.4	43	5	AAM50862		Beta-amyl
341	35	85.4	43	5	ABB78007		Amino aci
342	35	85.4	43	5	AAE26265	Aae26265	Human bet
343	35	85.4	43	6	AAO16064	Aao16064	Neurologi
344	35	85.4	43	6	ABG73456	Abg73456	Natural b
345	35	85.4	43	6	ABU08505		Human amy
346	35	85.4	43	6	ABP96145		Human Abe
				6	ABR39273	-	Human Amy
347	35	85.4	43				Amino aci
348	35	85.4	43	6	ABP97881		
349	35	85.4	43	6	ABU62720		Beta-amyl
350	35	85.4	43	7	ADC66003		Human A(b
351	35	85.4	45	2	AAR64169	Aar64169	Variant b

352	35	85.4	45	6	AAE35676	Aae35676	Human Abe
353	35	85.4	47	2	AAW81475	Aaw81475	Synthetic
354	35	85.4	48	4	AAB37523	Aab37523	Amyloid p
355	35	85.4	48	6	AAE35680	Aae35680	Human Abe
356	35	85.4	48	6	ABP97920	Abp97920	Amino aci
357	35	85.4	50	4	AAG65957	Aag65957	
358	35	85.4	52	2	AAR64166		Variant b
359	35	85.4	52	2	AAW81476		Synthetic
360	35	85.4	52	6	ABU08712		Amlyoid b
361	35	85.4	52	6	ABP97925		Amino aci
362	35	85.4	52	6	ABP97924		Amino aci
	35 35	85.4	52	6	ADA90299		Abeta ami
363			53	2	AAR55695	Aar55695	
364	35	85.4				Aar55696	=
365	35	85.4	53	2	AAR55696		Variant b
366	35	85.4	53	2	AAR64168		
367	35	85.4	53	3	AAY87944		Mammalian
368	35	85.4	53	6	ABU08708		Amlyoid b
369	35	85.4	53	6	AAO16342	Aao16342	
370	35	85.4	53	7	ADB61450		Amyloid b
371	35	85.4	54	3	AAB32126		Amyloid-b
372	35	85.4	54	6	AAO16345	Aao16345	
373	35	85.4	55	4	AAB11482		Human APP
374	35	85.4	55	4	AAE12903		Human bet
375	35	85.4	57	3	AAB10910		Human amy
376	35	85.4	58	2	AAW98001		Swedish-F
377	35	85.4	59	2	AAW05375	Aaw05375	Amyloid p
378	35	85.4	59	2	AAW70863		Beta-amyl
379	35	85.4	59	4	AAB84425	Aab84425	Partial s
380	35	85.4	59	7	ADB75160	Adb75160	Human bet
381	35	85.4	60	2	AAW49007	Aaw49007	Homo sapi
382	35	85.4	60	3	AAY69701	Aay69701	Beta-amyl
383	35	85.4	63	2	AAW42976	Aaw42976	Beta-amyl
384	35	85.4	63	2	AAW44747	Aaw44747	APP-REP 7
385	35	85.4	63	7	ADB33540	Adb33540	APP regio
386	35	85.4	63	7	ADB33534	Adb33534	APP regio
387	35	85.4	63	7	ADB33538	Adb33538	APP regio
388	35	85.4	63	7	ADB33537	Adb33537	APP regio
389	35	85.4	64	5	ABB81320	Abb81320	Amyloid p
390	35	85.4	67	2	AAW71377	Aaw71377	Peptide d
391		85.4		4	AAE09373		Human wil
392	35	85.4	70	4	AAE09374	Aae09374	Human APP
393	35	85.4	70	4	AAE09375	Aae09375	Human tru
394	35	85.4	70	4	AAU05015	Aau05015	Human amy
395	35	85.4	79	2	AAW53981		Human ALZ
396	35	85.4	82	5	AAU80960		Human amy
397	35	85.4	82	5	ABG94280		Amyloid b
398	35	85.4	82	5	ABG80592		Human amy
399	35	85.4	93	4	ABG19083		Novel hum
			97	1	AAP83152		Lambda SM
400	35 35	85.4 85.4	97 97	1	AAP81517	-	Deduced s
401	35 25	85.4					Beta-amyl
402	35	85.4	97	2	AAR37865		Sequence
403	35	85.4	99	2	AAR20329		Beta-amyl
404	35	85.4	99	2	AAR74696		
405	35	85.4	99	2	AAR74694		Beta-amyl
406	35	85.4	99	2	AAR64167		Variant b
407	35	85.4	99	2	AAY08606		Human bet
408	35	85.4	99	4	AAB11483	Aab11483	Human APP

409	35	85.4	99	5	ABB76945	Abb76945 Amyloid P
410	35	85.4	99	6	ABP97919	Abp97919 Amino aci
411	35	85.4	99	6	ABP97981	Abp97981 C99, the
412	35	85.4	100	2	AAR10024	Aar10024 Beta-amyl
		85.4	100	2	AAR37866	Aar37866 Full-leng
413	35					Aay51923 Transgeni
414	35	85.4	100	3	AAY51923	
415	35	85.4	100	3	AAB13015	Aab13015 Human amy
416	35	85.4	100	5	AAE14372	Aae14372 Amyloid p
417	35	85.4	100	5	AAE14373	Aae14373 Amyloid p
418	35	85.4	100	5	AAE14375	Aae14375 Amyloid p
419	35	85.4	100	5	AAE14371	Aae14371 Amyloid p
420	35	85.4	100	5	AAE14374	Aae14374 Amyloid p
421	35	85.4	100	6	ABP97921	Abp97921 Amino aci
422	35	85.4	103	2	AAR74697	Aar74697 Beta-amyl
423	35	85.4	103	2	AAR74698	Aar74698 Beta-amyl
		85.4		2		Aaw51317 Natural b
424	35		103		AAW51317	Aaw89372 Beta-amyl
425	35	85.4	103	2	AAW89372	,=
426	35	85.4	103	3	AAY56103	Aay56103 Beta amyl
427	35	85.4	103	4	AAE12509	Aae12509 Beta-amyl
428	35	85.4	103	5	ABG71002	Abg71002 Amyloid p
429	35	85.4	103	5	ABB05150	Abb05150 Beta amyl
430	35	85.4	103	6	ABG73457	Abg73457 Amyloid p
431	35	85.4	104	2	AAW51100	Aaw51100 Amino aci
432	35	85.4	108	1	AAP83154	Aap83154 Plasmid p
433	35	85.4	108	2	AAR37868	Aar37868 Beta-amyl
434	35	85.4	108	5	AAE14382	Aae14382 Gamma-sec
435	35	85.4	108	5	AAE14383	Aae14383 Gamma-sec
436	35	85.4	108	5	AAE14379	Aae14379 Gamma-sec
	35	85.4	108	5	AAE14380	Aae14380 Gamma-sec
437						Aae14381 Gamma-sec
438	35	85.4	108	5	AAE14381	Abp97923 Amino aci
439	35	85.4	108	6	ABP97923	<u>-</u>
440	35	85.4	112	2	AAR93556	Aar93556 Familial
441	35	85.4	115	2	AAW98000	Aaw98000 SwedishLo
442	35	85.4	115	2	AAW97999	Aaw97999 London-FA
443	35	85.4	115	2	AAW97997	Aaw97997 Swedish-F
444	35	85.4	116	3	AAY87823	Aay87823 Human APP
445	35	85.4	117	2	AAW51102	Aaw51102 Flag-amyl
446	35	85.4	117	3	AAY51925	Aay51925 Transgeni
447	35	85.4	117	4	AAE12896	Aae12896 Human rec
448	35	85.4	118	2	AAW50028	Aaw50028 APP C-ter
449	35	85.4	118	2	AAW50027	Aaw50027 APP C-ter
450	35	85.4	118	2	AAW50031	Aaw50031 APP C-ter
451	35	85.4	118	2	AAW50030	Aaw50030 APP C-ter
		85.4	118	2	AAW50029	Aaw50029 APP C-ter
452	35					Aaw96209 Amyloid p
453	35	85.4	118	2	AAW96209	Aaw50032 APP C-ter
454	35	85.4	120	2	AAW50032	
455	35	85.4	122	3	AAY97071	Aay97071 Beta-amyl
456	35	85.4	124	3	AAY96955	Aay96955 Beta-amyl
457	35	85.4	132	2	AAR65290	Aar65290 Rat beta
458	35	85.4	132	2	AAR65291	Aar65291 Human bet
459	35	85.4	247	5	AAE26274	Aae26274 Human bet
460	35	85.4	264	1	AAP90609	Aap90609 Sequence
461	35	85.4	264	1	AAP90497	Aap90497 Protein s
462	35	85.4	267	5	AAE26273	Aae26273 Human tPA
463	35	85.4	285	6	AA019900	Aao19900 BRI-Abeta
			285	6	AA019899	Aao19899 BRI-Abeta
464	35 35	85.4				Abp28084 Streptoco
465	35	85.4	295	5	ABP28084	Approvod Screpcoco

466	35	85.4	295	5	ABP29855	Abp29855 Streptoco
467	35	85.4	487	2	AAW26394	Aaw26394 Amyloid p
468	35	85.4	487	2	AAW26510	Aaw26510 Amyloid p
469	35	85.4	487	2	AAW42979	Aaw42979 Amyloid p
470	35	85.4	487	2	AAW44745	Aaw44745 APP-REP 7
471	35	85.4	492	2	AAR45229	Aar45229 APP-REP 7
472	35	85.4	492	2	AAW26393	Aaw26393 Amyloid p
				2		Aaw26509 Amyloid p
473	35	85.4	492		AAW26509	
474	35	85.4	492	2	AAW42978	Aaw42978 Amyloid p
475	35	85.4	492	2	AAW44744	Aaw44744 APP-REP 7
476	35	85.4	506	2	AAW61152	Aaw61152 Maltose b
477	35	85.4	506	2	AAY33742	Aay33742 MBP-APP (
478	35	85.4	506	4	AAB47258	Aab47258 MBP:APP C
479	35	85.4	534	6	ABB99605	Abb99605 Amino aci
480	35	85.4	537	2	AAR40114	Aar40114 APP-HCV-E
481	35	85.4	627	3	AAB10955	Aab10955 SEAP/huma
482	35	85.4	656	2	AAR58935	Aar58935 Amyloid p
483	35	85.4	670	5	ABB81499	Abb81499 Abeta42-H
484	35	85.4	676	2	AAR58936	Aar58936 Amyloid p
485	35	85.4	695	1	AAP81692	Aap81692 Sequence
486	35	85.4	695	2	AAR05166	Aar05166 Sequence
		85.4	695	2	AAR14046	Aar14046 Amyloid p
487	35			2		Aar26338 APP695. 3
488	35	85.4	695		AAR26338	
489	35	85.4	695	2	AAR58923	Aar58923 Mouse amy
490	35	85.4	695	2	AAR58920	Aar58920 Amyloid p
491	35	85.4	695	2	AAW19487	Aaw19487 APP695 mu
492	35	85.4	695	2	AAW19490	Aaw19490 APP695 mu
493	35	85.4	695	2	AAW19481	Aaw19481 APP695 mu
494	35	85.4	695	2	AAW19484	Aaw19484 APP695 mu
495	35	85.4	695	2	AAW19498	Aaw19498 APP695 mu
496	35	85.4	695	2	AAW19501	Aaw19501 APP695 mu
497	35	85.4	695	2	AAW19495	Aaw19495 APP695 mu
498	35	85.4	695	2	AAW19504	Aaw19504 APP695 mu
499	35	85.4	695	2	AAY20233	Aay20233 Human bet
500	35	85.4	695	2	AAY49690	Aay49690 Human bet
501	35	85.4	695	2	AAY07221	Aay07221 Amyloid p
502	35	85.4	695	3	AAY88435	Aay88435 Human APP
503	35	85.4	695	3	AAY88434	Aay88434 Human APP
504	35	85.4	695	3	AAY88436	Aay88436 Human APP
505	35	85.4				Aay44705 Human bet
				4	AAU07207	Aau07207 Human bet
506	35	85.4	695			Aau07207 Human bet
507	35	85.4	695	4	AAU07206	Aae10632 Human wil
508	35	85.4	695	4	AAE10632	
509	35	85.4	695	4	AAE10633	Aae10633 Human amy
510	35	85.4	695	4	AAE10634	Aae10634 Human amy
511	35	85.4	695	4	AAE06864	Aae06864 Human amy
512	35	85.4	695	4	AAE06862	Aae06862 Human wil
513	35	85.4	695	4	AAE06863	Aae06863 Human amy
514	35	85.4	695	4	AAE02584	Aae02584 Human amy
515	35	85.4	695	4	AAE02586	Aae02586 Human amy
516	35	85.4	695	4	AAE02585	Aae02585 Human amy
517	35	85.4	695	4	AAE03420	Aae03420 Human amy
518	35	85.4	695	4	AAU06608	Aau06608 Human Amy
519	35	85.4	695	4	AAU06607	Aau06607 Human Amy
520	35	85.4	695	4	AAU06606	Aau06606 Human Amy
	35	85.4	695	5	ABB78595	Abb78595 Human APP
521						Abb78594 Human APP
522	35	85.4	695	5	ABB78594	ADD/0334 Hullian Arr

523	35	85.4	695	5	ABB78593	Abb78593		
524	35	85.4	695	5	AAG68315	Aag68315		
525	35	85.4	695	5	ABG32721	Abg32721		
526	35	85.4	695	6	ABP97918	Abp97918	Amino a	ci
527	35	85.4	695	6	ABB99604	Abb99604	Amino a	ci
528	35	85.4	695	7	ADB87313	Adb87313	Human ar	ny
529	35	85.4	695	7	ADB87311	Adb87311		
530	35	85.4	695	7	ADB33519	Adb33519	Human Al	PP
531	35	85.4	695	7	ADC65997	Adc65997	Human Al	PP
532	35	85.4	697	3	AAY88429	Aay88429	Human Al	PP
533	35	85.4	697	3	AAY88430	Aay88430	Human Al	PΡ
534	35	85.4	697	3	AAY88428	Aay88428	Human Al	PΡ
535	35	85.4	697	4	AAU07208	Aau07208	Human be	et
536	35	85.4	697	4	AAU07210	Aau07210	Human be	et
537	35	85.4	697	4	AAU07209	Aau07209		
538	35	85.4	697	4	AAE10635	Aae10635		
539	35	85.4	697	4	AAE10637	Aae10637		
540	35	85.4	697	4	AAE10636	Aae10636		_
541	35	85.4	697	4	AAE06867	Aae06867		_
542	35	85.4	697	4	AAE06865	Aae06865		-
543	35	85.4	697	4	AAE06866	Aae06866		_
544	35	85.4	697	4	AAE02588	Aae02588		_
545	35	85.4	697	4	AAE02589	Aae02589		
546	35	85.4	697	4	AAE02587	Aae02587		_
547	35	85.4	697	4	AAU06609	Aau06609		-
	35	85.4	697	4	AAU06610	Aau06610		
548	35 35	85.4	697	4	AAU06611	Aau06611		_
549	35 35	85.4	697	5	ABB78597	Abb78597		
550	35 35	85.4	697	5	ABB78596	Abb78596		
551			697	5	ABB78598	Abb78598		
552	35 35	85.4 85.4	733	6	ABR43271	Abr43271		
553			740	7	ADB87314	Adb87314		
554	35	85.4	740	7	ADB87314 ADB87312	Adb87312		
555	35	85.4	751	1	ADB07312 AAP83150	Aap83150		
556	35	85.4		1		Aap94776		
557	35	85.4	751		AAP94776	Aar05718		_
558	35	85.4	751	2	AAR05718	Aar10022		
559	35	85.4	751	2	AAR10022			_
560	35	85.4	751	2	AAR20328	Aar20328	_	
561	35	85.4	751	2	AAR37862	Aar37862 Aaw19492		
562	35	85.4	751	2	AAW19492	Aaw19492 Aaw19489		
563	35	85.4	751	2	AAW19489			
564	35	85.4	751	2	AAW19486	Aaw19486		
565	35	85.4	751	2	AAW19483	Aaw19483		
566	35	85.4	751	2	AAW19505	Aaw19505		
567	35	85.4	751	2	AAW19502	Aaw19502		
568	35	85.4	751	2	AAW19496	Aaw19496		
569	35	85.4	751	2	AAW19499	Aaw19499		
570	35	85.4	751	2	AAY08615	Aay08615		
571	35	85.4	751	2	AAY08605	Aay08605		
572	35	85.4	751	4	AAE10649	Aae10649		_
573	35	85.4	751	4	AAE06894	Aae06894		-
574	35	85.4	751	4	AAE02601	Aae02601		
575	35	85.4	751	4	AAU06623	Aau06623	_	
576	35	85.4	751	5	ABB78610	Abb78610		
577	35	85.4	751	5	AAG68316	Aag68316		
578	35	85.4	751	5	ABG32722	Abg32722		
579	35	85.4	751	5	AAO18050	Aao18050	Amyloid	q

580	35	85.4	753	4	AAU07224		Aau07224	Human bet
581	35	85.4	753	4	AAE10651			Human amy
582	35	85.4	753	4	AAE06896			Human amy
583	35	85.4	753	4	AAE02603			Human amy
584	35	85.4	753	4	AAU06625			Human Amy
585	35	85.4	753	5	ABB78612			Human APP
586	35	85.4	754	2	AAR26339			APP751. 3
587	35	85.4	754	2	AAW96210			Amyloid p
588	35	85.4	768	5	AAU80959			Human amy
589	35	85.4	770	1	AAP94775			Novel amy
590	35	85.4	770	2	AAR05717			NAP gene
591	35	85.4	770	2	AAR26340		Aar26340	APP770. 3
592	35	85.4	770	2	AAR41546		Aar41546	Mutated A
593	35	85.4	770	2	AAR63442		Aar63442	Amyloid p
594	35	85.4	770	2	AAW19491		Aaw19491	APP770 mu
595	35	85.4	770	2	AAW19488		Aaw19488	APP770 mu
596	35	85.4	770	2	AAW19485		Aaw19485	APP770 mu
597	35	85.4	770	2	AAW19482		Aaw19482	APP770 mu
598	35	85.4	770	2	AAW19506			APP770 mu
599	35	85.4	770	2	AAW19497			APP770 mu
600	35	85.4	770	2	AAW19503			APP770 mu
601	35	85.4	770	2	AAW19500			APP770 mu
602	35	85.4	770	2	AAW40130			Human APP
603	35	85.4	770	2	AAW97996			Human amy
604	35	85.4	770	4	AAE11762			Human amy
605	35	85.4	770	4	AAE10648			Human amy
606	35	85.4	770	4	AAE06913			Human amy Human amy
607	35	85.4	770 770	4 4	AAE06912 AAE06893			Human amy
608 609	35 35	85.4 85.4	770	4	AAE02600			Human amy
610	35	85.4	770	4	AAU06622			Human par
611	35	85.4	770	5	ABG94279			Amyloid b
612	35	85.4	770	5	ABB78609		-	Human APP
613	35	85.4	770	5	ABG76936		Abg76936	Humanised
614	35	85.4	770	5	AAG68317			Human amy
615	35	85.4	770	5	ABB78008			Amino aci
616	35	85.4	770	5	ABG80591		_	Human amy
617	35	85.4	770	5	ABG32723		_	Human amy
618	35	85.4	770	6	ABP72693			Human amy
619	35	85.4	770	6	ABR43902			Beta-amyl
620	35	85.4	770	6	ABP97885			Amino aci
621	35	85.4	770	6	ABR61931			Human amy
622	35	85.4	772	4	AAU07223			Human bet
623	35	85.4	772	4	AAE10650			Human amy Human amy
624	35	85.4	772	4	AAE06895			Human amy
625	35	85.4	772	4	AAE02602			Human Amy
626	35	85.4	772 772	4	AAU06624 ABG19086			Novel hum
627	35 25	85.4 85.4	772	4 5	ABB78611		-	Human APP
628 629	35 35	85.4	777	4	ABG19089			Novel hum
630	35	85.4	783	7	ADB33513			Human APP
631	35	85.4	783	7	ADB33525			Human APP
632	35	85.4	783	7	ADB33531			Human APP
633	35	85.4	783	7	ADB33505			Human APP
634	35	85.4	783	7	ADB33503			Human APP
635	35	85.4	783	7	ADB33511		Adb33511	Human APP
636	35	85.4	941	7	ADB33507		Adb33507	Human APP
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637	35	85.4	941	7	ADB33515	Adb3	3515	Human APP
638	35	85.4	941	7	ADB33509			Human APP
639	35	85.4	941	7	ADB33533			Human APP
640	35	85.4	941	7	ADB33517			Human APP
641	35	85.4	941	7	ADB33527			Human APP
642	35	85.4	968	4	ABB63037			Drosophil
643	35	85.4	1024	5	AAU75873	*		APP-LacI
644	32	78.0	9	2	AAR45239			Mutant am
645	32	78.0	28	2	AAW01414			Beta/A4-a
646	32	78.0	28	4	AAB35600			Human clo
647	32	78.0	28	6	ABG72244			Mutant E2
648	32	78.0	35	4	AAB91830			Amyloid b
649	32	78.0	35	4	AAB91803			Amyloid b
650	32	78.0	40	2	AAW47232			Beta-amyl
651	32	78.0	42	6	ABP97887			Amino aci
652	32	78.0	53	2	AAR55697	-		Sequence
653	32	78.0	63	2	AAW26391			Amyloid p
654	32	78.0	63	2	AAW26511			Amyloid p
655	32	78.0	63	2	AAW42975			Beta-amyl
656	32	78.0	63	2	AAW44746			APP-REP 7
657	32	78.0	99	2	AAR74695	Aar7	4695	Beta-amyl
658	32	78.0	100	5	AAE14377	Aae1	4377	Amyloid p
659	32	78.0	108	5	AAE14385	Aae1	4385	Gamma-sec
660	32	78.0	184	6	ABU16515	Abu1	6515	Protein e
661	32	78.0	261	7	ABR62788	Abr6	2788	MRSA GTP
662	32	78.0	265	6	ABU43397	Abu4	3397	Protein e
663	32	78.0	268	6	ABM73194	Abm7	3194	Staphyloc
664	32	78.0	439	3	AAB01210	Aab0	1210	Corn puta
665	32	78.0	1142	4	ABG19749	Abg1	9749	Novel hum
666	31	75.6	6	6	ADA90176			Anti-Abet
667	31	75.6	7	[^] 6	ADA90156	Ada 9	0156	Anti-Abet
668	31	75.6	7	6	ADA90939			Solid-pha
669	31	75.6	8	3	AAY79939			Beta-amyl
670	31	75.6	9	6	ABU79049			Aggregati
671	31	75.6	9	7	ABW00183			Peptide #
672	31	75.6	10	4	AAB46230			Human APP
673	31	75.6	11	2	AAR60373			Beta-amyl
674	31	75.6	11	5	ABB04912			Human amy
675	31	75.6	12	3	AAB10958			Bovine AD
676	31	75.6	18	3	AAB10964			Beta-amyl
677	31	75.6	28	2	AAY39806	-		Beta-amyl
678	31	75.6	36	4	AAG75393			Human col
679	31	75.6	42	2	AAW67975			Fragment
680	31	75.6	42	6	ABP97888	_		Amino aci
681	31	75.6	42	6	ABP97886	-		Amino aci
682	31	75.6	49	2	AAR35087			Human amy
683	31	75.6	49	4	AAM14458			Peptide #
684	31	75.6	49	4	AAM13857			Peptide #
685	31	75.6	49	4	ABB32802			Peptide #
686	31	75.6	49	4	ABB33406			Peptide #
687	31	75.6	49	4	AAM26264			Peptide #
688	31	75.6	49	4	AAM26871			Peptide #
689	31	75.6	49	4	ABB27632			Human pep
690	31	75.6	49	4	ABB28231			Human pep
691	31	75.6	49	4	ABB18284			Protein #
692	31	75.6	49	4	ABB18865			Protein #
693	31	75.6	49	4	AAM66585	Aame	10000	Human bon

694	31	75.6	49	4	AAM65988	Aam65988 Human bon
695	31	75.6	49	4	AAM53609	Aam53609 Human bra
696	31	75.6	49	4	AAM54191	Aam54191 Human bra
697	31	75.6	49	4	ABG47654	Abg47654 Human liv
698	31	75.6	49	4	ABG48253	Abg48253 Human liv
699	31	75.6	49	4	AAM02185	Aam02185 Peptide #
700	31	75.6	49	4	AAM01600	Aam01600 Peptide #
701	31	75.6	49	5	ABG36237	Abg36237 Human pep
		75.6		5		Abg35636 Human pep
702	31		49		ABG35636	Abb41033 Peptide #
703	31	75.6	79	4	ABB41033	
704	31	75.6	79	4	AAM34806	Aam34806 Peptide #
705	31	75.6	79	4	ABB25109	Abb25109 Protein #
706	31	75.6	79	4	AAM74690	Aam74690 Human bon
707	31	75.6	79	4	AAM61888	Aam61888 Human bra
708	31	75.6	79	4	ABG56474	Abg56474 Human liv
709	31	75.6	79	5	ABG44503	Abg44503 Human pep
710	31	75.6	100	5	AAE14376	Aae14376 Amyloid p
711	31	75.6	104	4	AAE12897	Aae12897 Human rec
712	31	75.6	108	5	AAE14384	Aae14384 Gamma-sec
713	31	75.6	141	4	AAU19633	Aau19633 Human nov
714	31	75.6	141	5	ABP47853	Abp47853 Human pol
715	31	75.6	141	7	ADC10815	Adc10815 Human ext
716	31	75.6	164	2	AAY37480	Aay37480 Protein w
		75.6	170	6	ABU25390	Abu25390 Protein e
717	31			5	AAU02758	Aau02758 Human tum
718	31	75.6	195			
719	31	75.6	244	4	ABB67952	Abb67952 Drosophil
720	31	75.6	259	4	AAG92359	Aag92359 C glutami
721	31	75.6	291	5	ABB48134	Abb48134 Listeria
722	31	75.6	416	5	ABB81212	Abb81212 Human amy
723	31	75.6	471	3	AAB38627	Aab38627 Human sec
724	31	75.6	471	3	AAB38626	Aab38626 Gene 38 h
725	31	75.6	537	4	AAB95417	Aab95417 Human pro
726	31	75.6	579	2	AAR86406	Aar86406 Human mat
727	31	75.6	582	2	AAR86407	Aar86407 Human mat
728	31	75.6	582	2	AAR75648	Aar75648 Human pla
729	31	75.6	582	2	AAW52134	Aaw52134 Rabbit me
730	31	75.6	582	4	AAB84616	Aab84616 Amino aci
731	31	75.6	582	4	AAE10423	Aae10423 Human mat
732	31	75.6	582	5	AAU84294	Aau84294 Human end
733	31	75.6	582			Aae21037 Human mem
734	31	75.6	582	5	AAM50865	Aam50865 Matrix me
735	31	75.6	582	7	ADC15498	Adc15498 Human bas
				7	ADE64179	Ade64179 Human Pro
736	31	75.6	582			Aar62505 Amyloid p
737	31	75.6	770	2	AAR62505	Abp62957 Human pol
738	31	75.6	811	5	ABP62957	<u>-</u>
739	31	75.6	830	5	ABP62956	Abp62956 Human pol
740	31	75.6	896	5	ABJ10550	Abj10550 Human NOV
741	31	75.6	915	2	AAY13350	Aay13350 Amino aci
742	31	75.6	915	3	AAY95340	Aay95340 Human PRO
743	31	75.6	915	3	ADC78354	Adc78354 Human PRO
744	31	75.6	915	4	AAB80218	Aab80218 Human PRO
745	31	75.6	915	4	AAU12318	Aau12318 Human PRO
746	31	75.6	915	4	AAB53077	Aab53077 Human ang
747	31	75.6	915	6	ABU71596	Abu71596 Human PRO
748	31	75.6	915	6	AB017762	Abo17762 Novel hum
749	31	75.6	915	6	ABU71451	Abu71451 Human PRO
750	31	75.6	915	6	ABU81016	Abu81016 Human PRO
130	31	73.0	713	J	10001010	

751	31	75.6	915	6	ABU71897	Abu71897 Human sec
752	31	75.6	915	6	ABO01780	Abo01780 Novel hum
753	31	75.6	915	6	ABU66716	Abu66716 Human PRO
754	31	75.6	915	6	ABU54353	Abu54353 Human sec
755	31	75.6	915	6	ABO47368	Abo47368 Human sec
756	31	75.6	915	6	ABU59797	Abu59797 Novel sec
757	31	75.6	915	6	ABO24987	Abo24987 Human sec
758	31	75.6	915	6	ABU64505	Abu64505 Human sec
759	31	75.6	915	6	ABU67351	Abu67351 Human sec
		75.6	915	6	AB007331 AB014871	Abol4871 Human sec
760	31					Abu66992 Human sec
761	31	75.6	915	6	ABU66992	
762	31	75.6	915	6	ABU69628	Abu69628 Novel hum
763	31	75.6	915	6	ABO14810	Abol4810 Human sec
764	31	75.6	915	6	ADA45813	Ada45813 Novel hum
765	31	75.6	915	6	ADA76244	Ada76244 Human PRO
766	31	75.6	915	6	ADB29239	Adb29239 Human sec
767	31	75.6	915	6	ADA18894	Ada18894 Human PRO
768	31	75.6	915	6	ADA61517	Ada61517 Homo sapi
769	31	75.6	915	6	ADB19302	Adb19302 Novel hum
770	31	75.6	915	6	ADB27843	Adb27843 Human PRO
771	31	75.6	915	6	ADA86322	Ada86322 Novel hum
772	31	75.6	915	6	ADB15886	Adb15886 Human PRO
773	31	75.6	915	6	ADA47672	Ada47672 Human PRO
774	31	75.6	915	6	ADA18095	Ada18095 Human sec
775	31	75.6	915	6	AB032762	Abo32762 Human sec
776	31	75.6	915	6	ADA67467	Ada67467 Human PRO
777	31	75.6	915	6	ADB30474	Adb30474 Human PRO
778	31	75.6	915	6	ADA85770	Ada85770 Novel hum
779	31	75.6	915	6	ADA96982	Ada96982 Human PRO
780	31	75.6	915	6	ADA79286	Ada79286 Human PRO
781	31	75.6	915	6	ADA87425	Ada87425 Novel hum
782	31	75.6	915	6	ADB16627	Adb16627 Human PRO
783	31	75.6	915	6	ABO34822	Abo34822 Human PRO
784	31	75.6	915	6	ADA16070	Ada16070 Human sec
		75.6	915	6	ADA10070	Ada91719 Novel hum
785	31		915	6	ADB14782	Adb14782 Human PRO
786 787	31	75.6				Adb18743 Novel hum
787	31	75.6	915	6 6	ADB18743 ADA93958	Ada93958 Human PRO
788	31	75.6	915			Adb19854 Novel hum
789	31	75.6	915	6	ADB19854	
790	31	75.6	915		ADB13166	Adb13166 Human PRO Abo43295 Novel hum
791	31	75.6	915	6	ABO43295	
792	31	75.6	915	6	ADA74420	Ada74420 Human PRO
793	31	75.6	915	6	ADA42215	Ada42215 Human sec
794	31	75.6	915	6	ADB24653	Adb24653 Human PRO
795	31	75.6	915	6	ADA82177	Ada82177 Human PRO
796	31	75.6	915	6	ADA75140	Ada75140 Human PRO
797	31	75.6	915	6	ADA85218	Ada85218 Novel hum
798	31	75.6	915	6	ADA84666	Ada84666 Novel hum
799	31	75.6	915	6	ABO17500	Abo17500 Human PRO
800	31	75.6	915	6	ADB29922	Adb29922 Human PRO
801	31	75.6	915	6	ADA80450	Ada80450 Human PRO
802	31	75.6	915	6	ADA75692	Ada75692 Human PRO
803	31	75.6	915	6	ADA46917	Ada46917 Human PRO
804	31	75.6	915	6	ADB25213	Adb25213 Human PRO
805	31	75.6	915	6	ADA93389	Ada93389 Human PRO
806	31	75.6	915	6	ADB26739	Adb26739 Human PRO
807	31	75.6	915	6	ADB31026	Adb31026 Human PRO
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808	31	75.6	915	6	ADA60954	Ada60954	Homo s	api
809	31	75.6	915	6	ADB24101	Adb24101	Human	PRO
810	31	75.6	915	6	ADA96430	Ada96430	Human	PRO
811	31	75.6	915	6	ADA81002	Ada81002	Human	PRO
812		75.6	915	6	ADA95878	Ada95878	Human	PRO
813		75.6	915	6	ADB26187	Adb26187		
814		75.6	915	6	ADB21672	Adb21672		
815	31	75.6	915	7	ADA77451	Ada77451		
816	31	75.6	915	7	ADB18191	Adb18191		
817		75.6	915	7	ADA86874	Ada86874		
818		75.6	915	7	ADA16494	Ada16494		
819 ⁽⁾		75.6	915	7	ADA12923	Ada12923		
820		75.6	915	7	ADA41791	Ada41791		
821	31	75.6	915	7	ADA41751 ADA87977	Ada87977		
			915	7	ADA46365	Ada46365		
822	31	75.6				Ada17138		
823	31	75.6	915	7	ADA17138	Ada42641		
824	31	75.6	915	7	ADA42641	Adb28395		
825	31	75.6	915	7	ADB28395			
826		75.6	915	7	ADB28947	Adb28947		
827	31	75.6	915	7	ADA76899	Ada76899		
828	31	75.6	915	7	ADA88529	Ada88529		
829	31	75.6	915	7	ADA97534	Ada 97534		
830	31	75.6	915	7	ADB27291	Adb27291		
831	31	75.6	915	7	ADB22224	Adb22224		
832	31	75.6	915	7	AB017561	Abo17561		
833	31	75.6	915	7	ADA66915	Ada66915		
834	31	75.6	915	7	ADB22776	Adb22776		
835	31	75.6	915	7	ADB23549	Adb23549	Human	PRO
836	31	75.6	915	7	ADA92271	Ada92271	Novel	hum
837	31	75.6	915	7	ADB15334	Adb15334	Human	PRO
838	31	75.6	915	7	ADB38586	Adb38586	Novel	hum
839	31	75.6	915	7	ADB38034	Adb38034	Novel	hum
840	31	75.6	915	7	ADB66506	Adb66506	Novel	hum
841	31	75.6	915	7	ADB89586	Adb89586	Human	PRO
842	31	75.6	915	7	ADB90318	Adb90318	Human	PRO
843	31	75.6	915	7	ADB77560	Adb77560	Human	sec
844	31	75.6	915	7	ADB39419	Adb39419		
845	31	75.6	915	7	ADB74696	Adb74696		
846	31	75.6	915	7	ADB47042	Adb47042	Novel	hum
847	31	75.6	915			Adb86649		
848	31	75.6	915	7	ADB77254	Adb77254		
849	31	75.6	915	7	ADB34411	Adb34411		
850	31	75.6	915	7	ADB35515	Adb35515		
851	31	75.6	915	7	ADB33859	Adb33859		
852	31	75.6	915	7	ADB34963	Adb34963		
		75.6	915	7	ADB34903 ADB36067	Adb36067		
853	31		915			Adb46462		
854	31	75.6		7	ADB46462			
855	31	75.6	915	7	ADC28342	Adc28342		
856	31	75.6	915	7	ADC39542	Adc39542		
857	31	75.6	915	7	ADC40056	Adc40056		
858	31	75.6	915	7	ADC18884	Adc18884		
859	31	75.6	915	7	ADC34180	Adc34180		
860	31	75.6	915	7	ADC29235	Adc29235		
861	31	75.6	915	7	ADC28766	Adc28766		
862	31	75.6	915	7	ADC40651	Adc40651		
863	31	75.6	915	7	ADC19308	Adc19308		
864	31	75.6	915	7	ADC33756	Adc33756	Human	sec

	865	31	75.6	915	7	ADC12826	Adc12826 Human	sec
	866	31		915	7		Adc50335 Novel	
	867	31		915	7		Adc71882 Novel	
	868	31		915	7		Adc59861 Novel	
	869	31		915	7		Adc52868 Novel	
					7		Adc57222 Novel	
	870	31		915			Adc60413 Novel	
	871	31		915	7		Adc50888 Novel	
	872	31		915	7		Adc65415 Human	
	873	31		915	7			
	874	31	75.6	915	7		Adc54513 Novel	
	875	31	75.6	915	7		Adc53474 Novel	
	876	31	75.6	915	7		Adc58997 Novel	
	877	31	75.6	915	7		Adc55875 Novel	
	878	31	75.6	915	7		Adc58445 Novel	
	879	31	75.6	915	7		Adc12278 Human	
	880	31		915	7		Add03119 Novel	
	881	31	75.6	915	7		Adc90111 Novel	
	882	31	75.6	915	7		Adc69530 Human	
	883	31	75.6	915	7		Adc48419 Human	
	884	31	75.6	915	7	ADD09948	Add09948 Human	
	885	31	75.6	915	7	ADD04523	Add04523 Novel	
	886	31	75.6	915	7	ADC80479	Adc80479 Novel	hum
	887	31	75.6	915	7	ADD10986	Add10986 Human	PRO
	888	31	75.6	915	7	ADC47867	Adc47867 Human	PRO
	889	31	75.6	915	7	ADD04833	Add04833 Human	sec
	890	31	75.6	915	7	ADC79927	Adc79927 Novel	hum
	891	31	75.6	915	7	ADD09396	Add09396 Human	PRO
	892	31	75.6	915	7	ADD03839	Add03839 Human	sec
	893	31	75.6	915	7	ADD03415	Add03415 Human	sec
	894	31	75.6	915	7	ADD41109	Add41109 Novel	hum
	895	31	75.6	915	7	ADD52248	Add52248 Human	PRO
	896	31	75.6	915	7	ADD52988	Add52988 Human	PRO
,	897	31	75.6	915	7	ADD53540	Add53540 Novel	hum
	898	31	75.6	915	7	ADD51696	Add51696 Human	PRO
	899	31	75.6	915	7	ADD02495	Add02495 Human	PRO
	900	31	75.6	915	7	ADD01929	Add01929 Human	PRO
	901	31	75.6	915	7	ADD54111	Add54111 Novel	
	902	31	75.6	915	7	ADD92428	Add92428 Human	PRO
	903	31	75.6	915	7	ADD91324	Add91324 Human	PRO
	904	31	75.6	915	7	ADE03938	Ade03938 Human	
	905	31	75.6	915	7	ADE32235	Ade32235 Novel	
	906	31	75.6	915	7	ADE22167	Ade22167 Human	
	907	31	75.6	915	7	ADD79391	Add79391 Human	PRO
	908	31	75.6	915	7	ADE41927	Ade41927 Human	
	909	31	75.6	915	7	ADE17744	Adel7744 Human	
	910	31	75.6	915	7	ADD91876	Add91876 Human	
	911	31	75.6	915	7	ADE33339	Ade33339 Novel	
	912	31	75.6	915	7	ADE33391	Ade33891 Novel	
					7		Add79943 Human	
	913	31	75.6	915 915	7	ADD79943	Add92980 Human	
	914	31	75.6			ADD92980	Add92980 Human	
	915	31	75.6	915	7	ADE19400	Ade19400 Human	
	916	31	75.6	915	7	ADE10040		
	917	31	75.6	915	7	ADE18848	Ade18848 Human	
	918	31	75.6	915	7	ADE43044	Ade43044 Human	
	919	31.	75.6	915	7	ADD95833	Add95833 Human	
	920	31	75.6	915	7	ADE22719	Ade22719 Human	
	921	31	75.6	915	7	ADD78837	Add78837 Human	PRU

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922	31	75.6	915	7	ADE32787			Novel hum
923	31	75.6	915	7	ADE42479			Human PRO
924	31	75.6	915	7	ADD80495		Add80495	Human PRO
925	31	75.6	915	7	ADD89523		Add89523	Human PRO
926	31	75.6	915	7	ADE40807		Ade40807	Human PRO
927	31	75.6	915	7	ADE04606			Human PRO
928	31	75.6	915	8	ADC81031			Novel hum
929	31	75.6	915	8	ADE79112			Human sec
		75.6	915	8	ADD76479			Human PRO
930	31							
931	31	75.6	915	8	ADD87843			Human PRO
932	31	75.6	915	8	ADD86247			Human PRO
933	31	75.6	915	8	ADE79536			Human sec
934	31	75.6	915	8	ADE75695			Human PRO
935	31	75.6	915	8	ADE73212			Human sec
936	31	75.6	915	8	ADE23271			Human PRO
937	31	75.6	915	8	ADE23823		Ade23823	Human PRO
938	31	75.6	915	8	ADE24466		Ade24466	Human PRO
939	31	75.6	915	8	ADD87291		Add87291	Human PRO
940	31	75.6	915	8	ADE89157		Ade89157	Human PRO
941	31	75.6	915	8	ADE73747		Ade73747	Human sec
942	31	75.6	915	8	ADE18296		Ade18296	Human PRO
943	31	75.6	915	8	ADE88605			Human PRO
944	31	75.6	934	4	AAE03843			Human gen
								Human alb
945	31	75.6	934	5	ABG64542			Human pro
946	31	75.6	959	4	AAB20159			_
947	31	75.6	977	5	ABJ10549		-	Human NOV
948	31	75.6	983	4	AAE03877			Human gen
949	30	73.2	7	5	AAE29549			Amyloid b
950	30	73.2	8	5	AAE29553			Amyloid b
951	30	73.2	8	5	AAE29548			Amyloid b
952	30	73.2	9	5	AAE29552		Aae29552	Amyloid b
953	30	73.2	9	6	ABU79053		Abu79053	Aggregati
954	30	73.2	9	7	ABW00187		Abw00187	Peptide #
955	30	73.2	12	5	AAE29508		Aae29508	Amyloid b
956	30	73.2	12	5	AAE29517		Aae29517	Amyloid b
957	30	73.2	12	5	AAE29516			Amyloid b
958	30	73.2	12	5	AAE29507			Amyloid b
959	30	73.2	42	6	ABP97890			Amino aci
960	30	73.2	71	4	AAU14422			Human nov
961	30	73.2	153					E. faeciu
			179	4	ABG29049			Novel hum
962	30	73.2					_	Chlamydia
963	30	73.2	248	2	AAY35303		_	_
964	30	73.2	313	3	AAB59033			Breast an
965	30	73.2	420	2	AAW50282			Canine he
966	30	73.2	457	3	AAG51611		_	Arabidops
967	30	73.2	462	5	AAU96926			Sphingomo
968	30	73.2	462	7	ADD06104			Sphingomo
969	30	73.2	471	5	ABP99367		-	Arabidops
970	30	73.2	471	5	ABB92660		Abb92660	Herbicida
971	30	73.2	566	6	ABR53540		Abr53540	Protein s
972	30	73.2	570	4	ABB59776	•	Abb59776	Drosophil
973	30	73.2	582	2	AAW22499			Phaffia d
974	30	73.2	590	5	ABP29967			Streptoco
975	30	73.2	590	6	ABU46502		_	Protein e
	30	73.2	600	1	AAP91008			Prostagla
976							_	Streptoco
977	30	73.2	604	5	ABP25463		_	Protein e
978	30	73.2	664	6	ABU26628		ADUZOOZO	trocern e

979	30	73.2	755	5	AAU84267	Aau84267 Human end
980	30	73.2	795	4	AAB27229	Aab27229 Human EXM
981	30	73.2	880	3	AAY53621	Aay53621 Amino aci
982	30	73.2	887	6	ABU20576	Abu20576 Protein e
983	30	73.2	1184	4	AAB93276	Aab93276 Human pro
984	30	73.2	1516	4	AAM78705	Aam78705 Human pro
985	30	73.2	1516	4	AAM78702	Aam78702 Human pro
986	30	73.2	1516	6	ABU07411	Abu07411 Protein d
987	30	73.2	1780	7	ADE15980	Ade15980 G-coupled
988	29	70.7	6	2	AAW02327	Aaw02327 Beta-amyl
989	29	70.7	6	2	AAW89385	Aaw89385 Beta-amyl
990	29	70.7	6	5	ABG71027	Abg71027 Long form
991	29	70.7	6	5	ABB05173	Abb05173 Beta amyl
992	29	70.7	6	6	ADA90175	Ada90175 Anti-Abet
993	29	70.7	7	2	AAR88300	Aar88300 Non-amnes
994	29	70.7	7	2	AAR87921	Aar87921 Test pept
995	29	70.7	7	4	AAB67281	Aab67281 Residues
996	29	70.7	7	5	ABB04920	Abb04920 Human amy
997	29	70.7	7	6	ABB82630	Abb82630 Abeta fib
998	29	70.7	7	6	AAE35454	Aae35454 Abeta pep
999	29	70.7	7	6	AAE35453	Aae35453 Abeta pep
1000	29	70.7	7	6	ADA90938	Ada90938 Solid-pha

ALIGNMENTS

```
RESULT 1
AAE10662
    AAE10662 standard; peptide; 8 AA.
XX
    AAE10662;
AC
XX
DT
     10-DEC-2001 (first entry)
XX
     Human amyloid precursor protein substrate alpha-secretase peptide #1.
DE
XX
     Human; aspartyl protease 1; Aspl; amyloid precursor protein; APP;
KW
     Alzheimer's disease; AD; dementia; neurofibrillary tangle; gliosis;
KW
     amyloid plaque; neuronal loss; proteolytic; nootropic; neuroprotective;
KW
KW
     alpha-secretase.
XX
OS
     Homo sapiens.
XX
FH
     Kev
                     Location/Qualifiers
                     4. .5
FT
     Cleavage-site
     Misc-difference 8
FT
                     /note= "This residue is given as Val in the sequence
FT
                     shown as SEQ ID NO: 72 in pages 92 and 160 of the
FT
FT
                     specification"
XX
PN
     GB2357767-A.
XX
     04-JUL-2001.
PD
XX
     22-SEP-2000; 2000GB-00023315.
PF
XX
```

```
PR
     23-SEP-1999;
                    99US-00404133.
     23-SEP-1999;
                    99US-0155493P.
PR
     23-SEP-1999;
                    99WO-US020881.
PR
                    99US-00416901.
     13-OCT-1999;
PR
     06-DEC-1999;
                    99US-0169232P.
PR
XX
     (PHAA ) PHARMACIA & UPJOHN CO.
PA
XX
     Bienkowkski MJ, Gurney M;
ΡI
XX
     WPI; 2001-444208/48.
DR
XX
     Polypeptide comprising fragments of human aspartyl protease with amyloid
РΤ
     precursor protein processing activity and alpha-secretase activity, for
PT
     identifying modulators useful in treating Alzheimer's disease.
PT
XX
     Claim 10; Page 163; 187pp; English.
_{\mathrm{PS}}
XX
     The patent discloses human aspartyl protease 1 (hu-Asp1) or modified Asp1
CC
     proteins which lack transmembrane domain or amino terminal domain or
CC
     cytoplasmic domain and retains alpha-secretase activity and amyloid
CC
     protein precursor (APP) processing activity. The proteins of the
CC
     invention are useful for assaying hu-Asp1 alpha-secretase activity, which
CC
     in turn is useful for identifying modulators of hu-Asp1 alpha-secretase
CC
     activity, where modulators that increase hu-Aspl alpha-secretase activity
CC
     are useful for treating Alzheimer's disease (AD) which causes progressive
CC
     dementia with consequent formation of amyloid plaques, neurofibriklary
CC
     tangles, gliosis and neuronal loss. Hu-Asp1 protease substrate is useful
CC
     for assaying hu-Aspl proteolytic activity, by contacting hu-Aspl protein
CC
     with the substrate under acidic conditions and determining the level of
ĊC
     hu-Asp1 proteolytic activity. The present sequence is human amyloid
CC
     precursor protein (APP) substrate alpha-secretase peptide which is used
CC
     for determining the enzymatic activity of Asp-1 protein lacking
CC
     transmembrane domain (TM) and containing a (His)6 tag. Note: The present
CC
     sequence shown in page 163 of the specification is stated as being the
CC
     same as that shown in page 92 and page 160 of the specification. However
CC
     the sequence differs at the C-terminal end
CC
XX
SQ
     Sequence 8 AA;
                          100.0%; Score 41; DB 4; Length 8;
  Query Match
                          100.0%; Pred. No. 1.4e+06;
  Best Local Similarity
                                                                              0;
                               0; Mismatches
                                                   0; Indels
                                                                  0; Gaps
  Matches
            8; Conservative
            1 LVFFAEDF 8
Qy
              Db
            1 LVFFAEDF 8
RESULT 2
AAE02614
     AAE02614 standard; peptide; 8 AA.
XX
AC
     AAE02614;
XX
DT
     10-AUG-2001 (first entry)
XX
```

```
Human amyloid precursor protein substrate alpha-secretase peptide #1.
DE
XX
     Human; alpha-secretase; amyloid precursor protein; APP; therapy;
KW
     Alzheimer's disease; antialzheimer's; aspartyl protease 1; Asp1;
KW
     beta-secretase.
KW
XX
     Homo sapiens.
OS
XX
                     Location/Qualifiers
FH
     Key
FT
                     4. .5
     Cleavage-site
XX
PN
     WO200123533-A2.
XX
PD
     05-APR-2001.
XX
     22-SEP-2000; 2000WO-US026080.
PF
XX
     23-SEP-1999;
PR
                    99US-0155493P.
PR
     23-SEP-1999;
                    99WO-US020881.
PR
     13-OCT-1999;
                    99US-00416901.
PR
     06-DEC-1999;
                    99US-0169232P.
XX
     (PHAA ) PHARMACIA & UPJOHN CO.
PA
XX
PI
     Gurney M, Bienkowski MJ;
XX
     WPI; 2001-290516/30.
DR
XX
     Enzymes that cleave the alpha-secretase site of the amyloid precursor
PT
PT
     protein, useful for the treatment of Alzheimer's disease.
XX
     Claim 10; Page 98; 189pp; English.
PS
XX
     The present invention relates to enzymes for cleaving the alpha-
CC
     secretase site of the amyloid precursor protein (APP) and methods of
CC
     identifying those enzymes. The methods may be used to identify enzymes
CC
     that may be used to cleave the alpha-secretase cleavage site of the APP
CC
     protein. The enzymes may be used to treat or modulate the progress of
CC
     Alzheimer's disease. The present sequence is human amyloid precursor
CC
     protein (APP) substrate alpha-secretase peptide which is used for
CC
     determining the enzymatic activity of Asp-1 deltaTM (His)6 protein. Note:
CC
     The present sequence shown in page 98 of the specification is stated as
CC
     being the same as that shown in page 94 and page 188 of the
CC
     specification. However the sequence differs at the C-terminal end
CC
XX
SQ
     Sequence 8 AA;
  Query Match
                          100.0%; Score 41; DB 4; Length 8;
                                   Pred. No. 1.4e+06;
  Best Local Similarity
                          100.0%;
                                 0; Mismatches
                                                                              0;
  Matches
             8; Conservative
                                                    0; Indels
                                                                  0; Gaps
            1 LVFFAEDF 8
Qy
              11111111
Db
            1 LVFFAEDF 8
```

```
AAR08190 standard; peptide; 8 AA.
ID
XX
AC
    AAR08190;
XX
    25-MAR-2003
DT
                 (revised)
    09-JAN-2003 (revised)
DT
    13-FEB-1991
                 (first entry)
DT
XX
    Cerebrovascular amyloid peptide.
DE
XX
    Down's Syndrome; Alzheimer's; monoclonal antibody; amyloid plaques;
KW
K₩
    beta-amyloid precursor.
XX
OS
    Synthetic.
XX
    WO9012870-A.
PN
XX
PD
     01-NOV-1990.
XX
PF
    14-APR-1989;
                    89US-00338302.
XX
     14-APR-1989;
                    89US-00338302.
PR
XX
     (REME-) RES FOUND MENTAL HYGIENE INC.
PA
XX
     Kim KS, Wisniewski HM, Miller DL, Sapienza VJ, Eqbal IG;
PI
PΙ
     Chen CMJ;
XX
    WPI; 1990-348473/46.
DR
XX
     New monoclonal antibodies to peptide(s) associated with downs syndrome -
PT
     esp. to cerebrovascular amyloid protein, useful for diagnosis of the
PT
PT
     diseases in body fluids.
XX
PS
     Claim 9; Page 17; 25pp; English.
XX
     This synthetic peptide is elevated in individuals with Down's Syndrome
CC
     (DS) or Alzheimer's disease (AD). Monoclonal antibodies raised against it
CC
     are useful for the non-invasive diagnosis of DS and AD and in the study
CC
     of the beta-amyloid precursor protein. (Updated on 09-JAN-2003 to add
CC
CC
     missing OS field.) (Updated on 25-MAR-2003 to correct PA field.)
XX
SQ
     Sequence 8 AA;
                          85.4%; Score 35; DB 2; Length 8;
  Query Match
  Best Local Similarity
                          100.0%; Pred. No. 1.4e+06;
                                                                 0; Gaps
                                                                             0;
  Matches
            7; Conservative 0; Mismatches 0; Indels
            1 LVFFAED 7
Qу
              1 LVFFAED 7
RESULT 4
AAW32551
    AAW32551 standard; peptide; 8 AA.
```

```
XX
AC
     AAW32551;
XX
DT
     21-JAN-1998 (first entry)
XX
     Amyloidogenic sequence amyloid beta-peptide.
DE
XX
     Anti-amyloid peptide; iAbeta; abnormal protein folding inhibitor;
KW
     Alzheimer's disease; dementia; Down's syndrome; amyloidosis disorder;
KW
     human prion disease; Kuru; Creutzfeldt-Jakob disease;
KW
     Gerstmann-Straussler-Scheinker Syndrome; animal prion disease;
KW
     prion associated human neurodegenerative disease; scrapie;
KW
     spongiform encephalopathy; transmissible mink encephalopathy;
KW
     chronic wasting disease; mule; deer; elk; human.
KW
XX
     Homo sapiens.
OS
OS
     Synthetic.
XX
PN
     WO9639834-A1.
XX
PD
     19-DEC-1996.
XX
     06-JUN-1996;
                    96WO-US010220.
PF
XX
                    95US-00478326.
PR -
     07-JUN-1995;
     10-APR-1996;
                    96US-00630645.
PR
XX
     (UYNY ) UNIV NEW YORK STATE.
PA
XX
     Soto-Jara C, Baumann MH, Frangione B;
PI
XX
     WPI; 1997-051637/05.
DR
XX
PT
     New inhibitors of fibrillogenesis proteins or peptides - used for
PT
     preventing, treating or detecting amyloidosis disorders such as
PT
     Alzheimer's disease.
XX
     Disclosure; Fig 1A; 63pp; English.
PS
XX
     A method has been developed for the prevention or treatment of a disorder
CC
     or disease associated with the formation of amyloid or amyloid-like
CC
     deposits, involving the abnormal folding of a protein or peptide. The
CC
     method involves administering an inhibitory peptide which prevents the
CC
     abnormal folding or which dissolves existing amyloid or amyloid-like
CC
     deposits, where the peptide comprises a sequence of 3-15 amino acid
CC
     residues and has a hydrophobic cluster of at least 3 amino acids, where
CC
     at least one of the 3 amino acids is a beta-sheet blocking amino acid
CC
     residue selected from Pro, Gly, Asn and His. The present sequence
CC
     represents an amyloidogenic sequence, amyloid beta- peptide, which is
CC
     involved in the formation of several amyloid deposits. The inhibitory
CC
CC
     peptide is capable of associating with a structural determinant on the
     protein or peptide to structurally block and inhibit the abnormal folding
CC
     into amyloid or amyloid-like deposits. The method can be used for
CC
     preventing, treating or detecting e.g. Alzheimer's dementia or disease,
CC
     Down's syndrome, other amyloidosis disorders, human prion diseases such
CC
     as Kuru, Creutzfeldt-Jakob disease, Gerstmann- Straussler-Scheinker
CC
```

Syndrome, prion associated human neurodegenerative diseases or animal

CC

```
prion diseases such as scrapie, spongiform encephalopathy, transmissible
     mink encephalopathy and chronic wasting disease of mule deer and elk
CC
XX
     Sequence 8 AA;
SO
                          85.4%; Score 35; DB 2; Length 8;
  Query Match
                          100.0%; Pred. No. 1.4e+06;
  Best Local Similarity
            7; Conservative 0; Mismatches
                                                   0; Indels
                                                                  0; Gaps
                                                                              0;
            1 LVFFAED 7
Qу
              +1111111
            2 LVFFAED 8
Db
RESULT 5
AAE10663
     AAE10663 standard; peptide; 8 AA.
ID
XX
AC
     AAE10663;
XX
DT
     10-DEC-2001 (first entry)
XX
     Human amyloid precursor protein substrate alpha-secretase peptide #2.
DE
XX
     Human; aspartyl protease 1; Asp1; amyloid precursor protein; APP;
KW
KW
     Alzheimer's disease; AD; dementia; neurofibrillary tangle; gliosis;
     amyloid plaque; neuronal loss; proteolytic; nootropic; neuroprotective;
KW
KW
     alpha-secretase.
XX
OS
     Homo sapiens.
XX
                     Location/Qualifiers
FH
FT
     Cleavage-site
                     4. .5
XX
PN
     GB2357767-A.
XX
PD
     04-JUL-2001.
XX
PF
     22-SEP-2000; 2000GB-00023315.
XX
     23-SEP-1999;
                    99US-00404133.
PR
PR
     23-SEP-1999;
                    99US-0155493P.
     23-SEP-1999;
                    99WO-US020881.
PR
     13-OCT-1999;
                    99US-00416901.
PR
     06-DEC-1999;
                    99US-0169232P.
PR
XX
     (PHAA ) PHARMACIA & UPJOHN CO.
PA
XX
PΙ
     Bienkowkski MJ, Gurney M;
XX
DR
     WPI; 2001-444208/48.
XX
     Polypeptide comprising fragments of human aspartyl protease with amyloid
PT
     precursor protein processing activity and alpha-secretase activity, for
PT
     identifying modulators useful in treating Alzheimer's disease.
PT
XX
PS
     Claim 10; Page 163; 187pp; English.
```

```
The patent discloses human aspartyl protease 1 (hu-Asp1) or modified Asp1
CC
     proteins which lack transmembrane domain or amino terminal domain or
CC
     cytoplasmic domain and retains alpha-secretase activity and amyloid
CC
     protein precursor (APP) processing activity. The proteins of the
CC
     invention are useful for assaying hu-Asp1 alpha-secretase activity, which
CC
     in turn is useful for identifying modulators of hu-Aspl alpha-secretase
CC
     activity, where modulators that increase hu-Aspl alpha-secretase activity
CC
     are useful for treating Alzheimer's disease (AD) which causes progressive
CC
     dementia with consequent formation of amyloid plaques, neurofibrillary
CC
     tangles, gliosis and neuronal loss. Hu-Asp1 protease substrate is useful
CC
     for assaying hu-Aspl proteolytic activity, by contacting hu-Aspl protein
CC
     with the substrate under acidic conditions and determining the level of
CC
     hu-Aspl proteolytic activity. The present sequence is human amyloid
CC
     precursor protein (APP) substrate alpha-secretase peptide which is used
CC
     for determining the enzymatic activity of Asp-1 protein lacking
CC
     transmembrane domain (TM) and containing a (His)6 tag
CC
XX
SO
     Sequence 8 AA;
                          85.4%; Score 35; DB 4; Length 8;
  Query Match
                          100.0%; Pred. No. 1.4e+06;
  Best Local Similarity
                               0; Mismatches
                                                   0; Indels
                                                                 0; Gaps
                                                                             0;
            7; Conservative
  Matches
            1 LVFFAED 7
Qу
              2 LVFFAED 8
Db
RESULT 6
AAE02615
     AAE02615 standard; peptide; 8 AA.
XX
AC
     AAE02615;
XX
DT
     10-AUG-2001 (first entry)
XX
     Human amyloid precursor protein substrate alpha-secretase peptide #2.
DE
XX
     Human; alpha-secretase; amyloid precursor protein; APP; therapy;
KW
     Alzheimer's disease; antialzheimer's; aspartyl protease 1; Asp1;
KW
KW
     beta-secretase.
XX
OS
     Homo sapiens.
XX
FΗ
     Kev
                     Location/Qualifiers
FT
     Cleavage-site
                     4. .5
XX
PN
     WO200123533-A2.
XX
PD
     05-APR-2001.
XX
     22-SEP-2000; 2000WO-US026080.
PF
XX
                    99US-0155493P.
PR
     23-SEP-1999;
                    99WO-US020881.
PR
     23-SEP-1999;
PR
     13-OCT-1999;
                    99US-00416901.
```

XX

```
PR
     06-DEC-1999;
                    99US-0169232P.
XX
PΑ
     (PHAA ) PHARMACIA & UPJOHN CO.
XX
PΙ
     Gurney M,
               Bienkowski MJ;
XX
     WPI; 2001-290516/30.
DR
XX
     Enzymes that cleave the alpha-secretase site of the amyloid precursor
PT
     protein, useful for the treatment of Alzheimer's disease.
PT
XX
     Claim 10; Page 98; 189pp; English.
PS
XX
     The present invention relates to enzymes for cleaving the alpha-
CC
     secretase site of the amyloid precursor protein (APP) and methods of
CC
     identifying those enzymes. The methods may be used to identify enzymes
CC
     that may be used to cleave the alpha-secretase cleavage site of the APP
CC
     protein. The enzymes may be used to treat or modulate the progress of
CC
     Alzheimer's disease. The present sequence is human amyloid precursor
CC
     protein (APP) substrate alpha-secretase peptide which is used for
CC
     determining the enzymatic activity of Asp-1 deltaTM (His)6 protein
CC
XX
SQ
     Sequence 8 AA;
                          85.4%; Score 35; DB 4; Length 8;
  Query Match
  Best Local Similarity
                          100.0%; Pred. No. 1.4e+06;
             7; Conservative 0; Mismatches
                                                   0; Indels
                                                                  0; Gaps
                                                                              0;
  Matches
            1 LVFFAED 7
Qу
              2 LVFFAED 8
Db
RESULT 7
ABB78624
     ABB78624 standard; peptide; 8 AA.
ID
XX
AC
     ABB78624;
XX
DT
     16-JUL-2002 (first entry)
XX
     Human alpha secretase (Abeta12-28) peptide SEQ ID NO:73.
DE
XX
     Human; Asp-1; Asp-2; aspartyl protease; Alzheimer's disease; proteolytic.
KW
XX
OS
     Homo sapiens.
XX
PN
     GB2367060-A.
XX
PD
     27-MAR-2002.
XX
     29-OCT-2001; 2001GB-00025934.
PF
XX
                    99US-00404133.
PR
     23-SEP-1999;
                    99US-0155493P.
PR
     23-SEP-1999;
                    99WO-US020881.
PR
     23-SEP-1999;
PR
     13-OCT-1999;
                    99US-00416901.
```

```
06-DEC-1999;
                    99US-0169232P.
     22-SEP-2000; 2000GB-00023315.
PR
XX
     (PHAA ) PHARMACIA & UPJOHN CO.
PΑ
XX
     Bienkowkski MJ, Gurney M;
PΙ
XX
     WPI; 2002-397167/43.
DR
XX
     Human aspartyl protease 1 substrates useful in assays to detect aspartyl
PT
     protease activity, e.g. for the diagnosis of Alzheimer's disease.
PT
XX
     Example 15; Page 92; 182pp; English.
PS
XX
     The present invention describes a human aspartyl protease 1 (hu-Asp1)
CC
     substrate (I) which comprises a peptide of no more than 50 amino acids,
CC
     and which comprises the 8 amino acid sequence Gly-Leu-Ala-Leu-Ala-Leu-
CC
     Glu-Pro. Also described are: (1) a method (II) for assaying hu-Aspl
CC
     proteolytic activity, comprising: (a) contacting a hu-Aspl protein with
CC
     (I) under acidic conditions; and (b) determining the level of hu-Aspl
CC
     proteolytic activity; (2) a purified polynucleotide (III) comprising a
CC
     nucleotide sequence that hybridises under stringent conditions to the non
CC
     -coding strand complementary to a defined 1804 nucleotide sequence (see
CC
     ABL52456) where the nucleotide sequence encodes a polypeptide having Aspl
CC
     proteolytic activity and lacks nucleotides encoding a transmembrane
CC
     domain); (3) a purified polynucleotide (III') comprising a sequence that
CC
     hybridises under stringent conditions to (III) (the nucleotide sequence
CC
     encodes a polypeptide further lacking a pro-peptide domain corresponding
CC
CC
     to amino acids 23-62 of hu-Asp1 (see ABB78589)); (4) a vector (IV)
     comprising (III) or (III'); and (5) a host cell (V) transformed or
CC
     transfected with (III), (III') and/or (IV). The hu-Aspl protease
CC
     substrate (I) may be used as an enzyme substrate in assays to detect
CC
     aspartyl protease activity, (II) and therefore diagnose diseases
CC
     associated with aberrant hu-Asp1 expression and activity such as
CC
     Alzheimer's disease. Hu-Aspl has been localised to chromosome 21, while
CC
     hu-Asp2 has been localised to chromosome 11q23.3-24.1. The present
CC
     sequence represents a human alpha secretase peptide, which is used in an
CC
CC
     example from the present invention
XX
     Sequence 8 AA;
SO
                          85.4%; Score 35; DB 5; Length 8;
  Query Match
                          100.0%; Pred. No. 1.4e+06;
  Best Local Similarity
                               0; Mismatches
                                                                     Gaps
                                                                              0;
                                                 0; Indels
             7; Conservative
  Matches
            1 LVFFAED 7
Qу
              Db
            2 LVFFAED 8
RESULT 8
ABB78623
ID
     ABB78623 standard; peptide; 8 AA.
XX
     ABB78623;
AC
XX
DT
     16-JUL-2002 (first entry)
```

```
XX
     Human alpha secretase (Abeta12-28) peptide SEQ ID NO:72.
DE
XX
     Human; Asp-1; Asp-2; aspartyl protease; Alzheimer's disease; proteolytic.
KW
XX
OS
     Homo sapiens.
XX
ΡN
     GB2367060-A.
XX
PD
     27-MAR-2002.
XX
PF
     29-OCT-2001; 2001GB-00025934.
XX
                    99US-00404133.
PR
     23-SEP-1999;
                    99US-0155493P.
PR
     23-SEP-1999;
     23-SEP-1999;
                    99WO-US020881.
PR
     13-OCT-1999;
                    99US-00416901.
PR
PR
     06-DEC-1999;
                    99US-0169232P.
     22-SEP-2000; 2000GB-00023315.
PR
XX
PΑ
     (PHAA ) PHARMACIA & UPJOHN CO.
XX
PI
     Bienkowkski MJ, Gurney M;
XX
     WPI; 2002-397167/43.
DR
XX
     Human aspartyl protease 1 substrates useful in assays to detect aspartyl
PT
     protease activity, e.g. for the diagnosis of Alzheimer's disease.
PT
XX
PS
     Example 15; Page 92; 182pp; English.
XX
     The present invention describes a human aspartyl protease 1 (hu-Asp1)
CC
CC
     substrate (I) which comprises a peptide of no more than 50 amino acids,
CC
     and which comprises the 8 amino acid sequence Gly-Leu-Ala-Leu-Ala-Leu-
CC
     Glu-Pro. Also described are: (1) a method (II) for assaying hu-Asp1
     proteolytic activity, comprising: (a) contacting a hu-Aspl protein with
CC
     (I) under acidic conditions; and (b) determining the level of hu-Aspl
CC
CC
     proteolytic activity; (2) a purified polynucleotide (III) comprising a
     nucleotide sequence that hybridises under stringent conditions to the non
CC
     -coding strand complementary to a defined 1804 nucleotide sequence (see
CC
     ABL52456) where the nucleotide sequence encodes a polypeptide having Asp1
CC
     proteolytic activity and lacks nucleotides encoding a transmembrane
CC
CC
     domain); (3) a purified polynucleotide (III') comprising a sequence that
     hybridises under stringent conditions to (III) (the nucleotide sequence
CC
     encodes a polypeptide further lacking a pro-peptide domain corresponding
CC
CC
     to amino acids 23-62 of hu-Asp1 (see ABB78589)); (4) a vector (IV)
     comprising (III) or (III'); and (5) a host cell (V) transformed or
CC
     transfected with (III), (III') and/or (IV). The hu-Asp1 protease
CC
     substrate (I) may be used as an enzyme substrate in assays to detect
CC
CC
     aspartyl protease activity, (II) and therefore diagnose diseases
CC
     associated with aberrant hu-Aspl expression and activity such as
     Alzheimer's disease. Hu-Aspl has been localised to chromosome 21, while
CC
     hu-Asp2 has been localised to chromosome 11q23.3-24.1. The present
CC
     sequence represents a human alpha secretase peptide, which is used in an
CC
     example from the present invention
CC
```

XX SQ

```
85.4%; Score 35; DB 5; Length 8;
 Query Match
                         100.0%; Pred. No. 1.4e+06;
 Best Local Similarity
                                                                 0; Gaps
            7; Conservative 0; Mismatches 0; Indels
 Matches
            1 LVFFAED 7
Qу
              Db
           1 LVFFAED 7
RESULT 9
ABU09765
     ABU09765 standard; peptide; 8 AA.
XX
     ABU09765;
AC
XX
     17-JUN-2003 (first entry)
DT
XX
DE
     Amyloidogenic Amyloid beta-peptide #1.
XX
KW
     Amyloid formation; amyloid-like deposit; Alzheimer's disease;
     pathological beta-sheet-rich conformation; Down's syndrome;
KW
     amyloidosis disorder; human prion disease; kuru; CJD;
KW
     Creutzfeldt-Jakob disease; Gerstmann-Straussler-Scheinker syndrome; GSS;
KW
     prion associated human neurodegenerative disease; animal prion disease;
KW
     scrapie; spongiform encephalopathy; transmissible mink encephalopathy;
KW
     chronic wasting disease.
KW
XX
OS
     Homo sapiens.
XX
     US6462171-B1.
PN
XX
PD
     08-OCT-2002.
XX
PF
     12-DEC-1996;
                    96US-00766596.
XX
PR
     07-JUN-1995;
                    95US-00478326.
PR
     10-APR-1996;
                    96US-00630645.
XX
     (UYNY ) UNIV NEW YORK STATE.
PA
XX
PT
     Soto-Jara C, Baumann MH, Frangione B;
XX
     WPI; 2003-379012/36.
DR
XX
РΤ
     Novel inhibitory peptides which inhibit and structurally block abnormal
     folding of protein into amyloid or amyloid-like deposit and into
PT
     pathological beta-sheet rich conformation, useful for treating
PT
PT
     Alzheimer's disease.
XX
PS
     Example 1; Fig 1A; 51pp; English.
XX
     The invention describes an isolated inhibitory peptide (I) which
CC
     interacts with a hydrophobic beta-sheet forming cluster of amino acid
CC
     residues on a protein or peptide for amyloid or amyloid-like deposit
CC
     formation, and inhibits or structurally blocks the abnormal folding of
CC
     proteins and peptides into amyloid or amyloid-like deposits and into
CC
```

0:

```
pathological beta-sheet-rich conformation. (I) is useful for disorders or
     diseases associated with abnormal protein folding into amyloid or amyloid
CC
     -like deposits or into pathological beta-sheet-rich precursors of such
CC
     deposits, such as Alzheimer's disease, Down's syndrome, other amyloidosis
CC
     disorders, human prion diseases, such as kuru, Creutzfeldt-Jakob disease
CC
     (CJD), Gerstmann-Straussler-Scheinker syndrome (GSS), prion associated
CC
     human neurodegenerative diseases as well as animal prion diseases such as
CC
     scrapie, spongiform encephalopathy, transmissible mink encephalopathy and
CC
     chronic wasting disease of mule deer and elk. (I) is also useful for
CC
     detecting and diagnosing the presence or absence of amyloid or amyloid-
CC
     like deposits in vivo and its precursors. This is the amino acid sequence
CC
     of peptide associated with the inhibition of amyloid or amyloid like
CC
CC
     deposits
XX
SO
     Sequence 8 AA;
                          85.4%; Score 35; DB 6; Length 8;
 Query Match
                          100.0%; Pred. No. 1.4e+06;
 Best Local Similarity
                                                                              0:
             7; Conservative
                               0; Mismatches
                                                   0; Indels
                                                                  0; Gaps
 Matches
            1 LVFFAED 7
Qу
              1111111
            2 LVFFAED 8
Db
RESULT 10
ABR61959
     ABR61959 standard; protein; 8 AA.
XX
AC
     ABR61959;
XX
     12-SEP-2003 (first entry)
DT
XX
DΕ
     Human amyloid precursor protein (APP) fragment.
XX
     Memapsin 1; nootropic; neuroprotective; memapsin 2; beta secretase;
KW
     beta-amyloid protein; Alzheimer's disease; amyloid precursor protein;
KW
KW
     APP; human.
XX
OS
     Homo sapiens.
XX
PN
     WO2003039454-A2.
XX
     15-MAY-2003.
PD
XX
PF
     23-OCT-2002; 2002WO-US034324.
XX
PR
     23-OCT-2001; 2001US-0335952P.
PR
     27-NOV-2001; 2001US-0333545P.
PR
     14-JAN-2002; 2002US-0348464P.
PR
     14-JAN-2002; 2002US-0348615P.
     20-JUN-2002; 2002US-0390804P.
PR
     19-JUL-2002; 2002US-0397557P.
PR
PR
     19-JUL-2002; 2002US-0397619P.
XX
PΑ
     (OKLA-) OKLAHOMA MEDICAL RES FOUND.
PΑ
     (UNII ) UNIV ILLINOIS FOUND.
```

```
XX
                Tang J, Bilcer G, Chang W, Hong L, Koelsch G, Loy J;
ΡI
PΙ
     Turner RT;
XX
     WPI; 2003-541410/51.
DR
XX
     New peptide compounds are memapsin beta secretase inhibitors used for
PT
     treating Alzheimer's disease.
PT
XX
     Example 2; Page 156; 407pp; English.
PS
XX
     The invention relates to peptide compounds of specified formula. The
CC
     compounds exhibit memapsin 2-beta secretase inhibitory activity relative
CC
     to memapsin 1-beta secretase and reduce the accumulation of beta-amyloid
CC
     protein. The compounds can be used for treating Alzheimer's disease. The
CC
     present sequence represents a human amyloid precursor protein (APP)
CC
     fragment where hydolysis by memapsin takes place
CC
XX
SQ
     Sequence 8 AA;
                          85.4%; Score 35; DB 6; Length 8; 100.0%; Pred. No. 1.4e+06;
  Query Match
  Best Local Similarity
             7; Conservative 0; Mismatches
                                                  0; Indels
                                                                  0; Gaps
                                                                               0;
  Matches
            1 LVFFAED 7
Qy
              Db
            2 LVFFAED 8
RESULT 11
ABW00134
ID
     ABW00134 standard; peptide; 8 AA.
XX
AC
     ABW00134;
XX
DT
     15-JAN-2004 (first entry)
XX
DE
     Beta-amyloid peptide.
XX
     Amyloid-like fibril deposit; prion related encephalopathy; gene therapy;
KW
     Alzheimer's disease; beta-amyloid.
KW
XX
     Unidentified.
OS
XX
PN
     US2003087407-A1.
XX
PD
     08-MAY-2003.
XX
PF
     06-SEP-2002; 2002US-00235483.
XX
     07-JUN-1995;
PR
                    95US-00478326.
                    96US-00630645.
PR
     10-APR-1996;
                    96US-00766596.
PR
     12-DEC-1996;
XX
     (UYNY ) UNIV NEW YORK STATE.
PΑ
XX
PI
     Soto-Jara C, Baumann MH, Frangione B;
```

```
XX
DR
     WPI; 2003-616149/58.
XX
     New inhibitory peptide, useful for preparing a composition for
PT
     diagnosing, preventing or treating disorders associated with amyloid-like
PT
     fibril deposits, e.g. Alzheimer's disease, or prion related
PT
     encephalopathies.
PT
XX
     Example 1; Fig 1A; 52pp; English.
PS
XX
     The invention relates to inhibitory peptide comprising a portion of at
CC
     least three amino acid residues and a sequence predicted not to adopt a
CC
     beta-sheet structure that associates with a hydrophobic beta-sheet
CC
     cluster on a protein or peptide involved in the abnormal folding into a
CC
     beta-sheet structure, to structurally block the abnormal folding of the
CC
     protein or peptide. The inhibitory peptide is useful for preparing a
CC
     composition for preventing, treating or detecting disorders or diseases
CC
     associated with amyloid-like fibril deposits e.g. Alzheimer's disease and
CC
     prion related encephalopathies. The invention is also useful in gene
CC
     therapy. The present sequence is beta-amyloid peptide. This peptide is
CC
     involved in the formation of several amyloid deposits
CC
XX
SQ
     Sequence 8 AA;
                          85.4%; Score 35; DB 7; Length 8;
  Query Match
                          100.0%; Pred. No. 1.4e+06;
  Best Local Similarity
            7; Conservative
                                0; Mismatches
                                                   0; Indels
                                                                  0; Gaps
                                                                              0;
  Matches
            1 LVFFAED 7
Qу
              111111
            2 LVFFAED 8
Db
RESULT 12
ABU79063
     ABU79063 standard; peptide; 9 AA.
ID
XX
AC
     ABU79063;
XX
DT
     17-JUN-2003 (first entry)
XX
DE
     Aggregation blocking peptide #15.
XX
     Amyloid formation; amyloid-like deposit; Alzheimer's disease;
KW
     pathological beta-sheet-rich conformation; Down's syndrome;
KW
     amyloidosis disorder; human prion disease; kuru; CJD;
KW
     Creutzfeldt-Jakob disease; Gerstmann-Straussler-Scheinker syndrome; GSS;
KW
     prion associated human neurodegenerative disease; animal prion disease;
KW
     scrapie; spongiform encephalopathy; transmissible mink encephalopathy;
KW
KW
     chronic wasting disease.
XX
OS
     Unidentified.
XX
PN
     US6462171-B1.
XX
PD
     08-OCT-2002.
XX
```

```
12-DEC-1996;
                    96US-00766596.
PF
XX
                    95US-00478326.
     07-JUN-1995;
PR
                    96US-00630645.
PR
     10-APR-1996;
XX
     (UYNY ) UNIV NEW YORK STATE.
PΑ
XX
     Soto-Jara C, Baumann MH, Frangione B;
PI
XX
     WPI; 2003-379012/36.
DR
XX
     Novel inhibitory peptides which inhibit and structurally block abnormal
PT
     folding of protein into amyloid or amyloid-like deposit and into
PT
     pathological beta-sheet rich conformation, useful for treating
PΤ
     Alzheimer's disease.
PT
XX
     Disclosure; Col 51-52; 51pp; English.
PS
XX
     The invention describes an isolated inhibitory peptide (I) which
CC
     interacts with a hydrophobic beta-sheet forming cluster of amino acid
CC
     residues on a protein or peptide for amyloid or amyloid-like deposit
CC
     formation, and inhibits or structurally blocks the abnormal folding of
CC
     proteins and peptides into amyloid or amyloid-like deposits and into
CC
     pathological beta-sheet-rich conformation. (I) is useful for disorders or
CC
     diseases associated with abnormal protein folding into amyloid or amyloid
CC
     -like deposits or into pathological beta-sheet-rich precursors of such
CC
     deposits, such as Alzheimer's disease, Down's syndrome, other amyloidosis
CC
     disorders, human prion diseases, such as kuru, Creutzfeldt-Jakob disease
CC
     (CJD), Gerstmann-Straussler-Scheinker syndrome (GSS), prion associated
CC
     human neurodegenerative diseases as well as animal prion diseases such as
CC
     scrapie, spongiform encephalopathy, transmissible mink encephalopathy and
CC
     chronic wasting disease of mule deer and elk. (I) is also useful for
CC
     detecting and diagnosing the presence or absence of amyloid or amyloid-
CC
     like deposits in vivo and its precursors. This is the amino acid sequence
CC
     of peptide associated with the inhibition of amyloid or amyloid like
CC
CC
     deposits
XX
SO
     Sequence 9 AA;
                          85.4%; Score 35; DB 6; Length 9;
  Query Match
                          100.0%; Pred. No. 1.4e+06;
  Best Local Similarity
                                                                             0;
                                                                 0; Gaps
                                                 0; Indels
  Matches
             7: Conservative 0; Mismatches
            1 LVFFAED 7
Qу
              Dh
            3 LVFFAED 9
RESULT 13
ABW00197
ID
     ABW00197 standard; peptide; 9 AA.
XX
AC
     ABW00197;
XX
     15-JAN-2004 (first entry)
DT
XX
DE
     Peptide #15 used in the invention.
```

```
XX
     Amyloid-like fibril deposit; prion related encephalopathy; gene therapy;
KW
     Alzheimer's disease.
KW
XX
     Unidentified.
OS
XX
     US2003087407-A1.
PN
XX
     08-MAY-2003.
PD
XX
     06-SEP-2002; 2002US-00235483.
PF
XX
     07-JUN-1995;
                    95US-00478326.
PR
                    96US-00630645.
     10-APR-1996;
PR
                    96US-00766596.
     12-DEC-1996;
PR
XX
     (UYNY ) UNIV NEW YORK STATE.
PΑ
XX
     Soto-Jara C, Baumann MH, Frangione B;
PΙ
XX
DR
     WPI; 2003-616149/58.
XX
     New inhibitory peptide, useful for preparing a composition for
PT
     diagnosing, preventing or treating disorders associated with amyloid-like
PT
     fibril deposits, e.g. Alzheimer's disease, or prion related
PT
PT
     encephalopathies.
XX
     Claim 1; Page 28; 52pp; English.
PS
XX
     The invention relates to inhibitory peptide comprising a portion of at
CC
     least three amino acid residues and a sequence predicted not to adopt a
CC
     beta-sheet structure that associates with a hydrophobic beta-sheet
CC
     cluster on a protein or peptide involved in the abnormal folding into a
CC
     beta-sheet structure, to structurally block the abnormal folding of the
CC
     protein or peptide. The inhibitory peptide is useful for preparing a
CC
     composition for preventing, treating or detecting disorders or diseases
CC
     associated with amyloid-like fibril deposits e.g. Alzheimer's disease and
CC
     prion related encephalopathies. The invention is also useful in gene
CC
     therapy. The present sequence is a peptide used in the invention
CC
XX
SO
     Sequence 9 AA;
                          85.4%; Score 35; DB 7; Length 9;
  Query Match
                          100.0%; Pred. No. 1.4e+06;
  Best Local Similarity
                                                                  0; Gaps
                                                                              0;
                                0; Mismatches
                                                    0; Indels
            7; Conservative
            1 LVFFAED 7
Qу
              111111
            3 LVFFAED 9
RESULT 14
AAY79938
     AAY79938 standard; peptide; 10 AA.
ID
XX
AC
     AAY79938;
XX
```

```
11-MAY-2000 (first entry)
DT
XX
DΕ
     Beta-amyloid recognition peptide SEQ ID NO:3.
XX
     Beta-amyloid; inhibitor; recognition element; hybrid; aggregation;
KW
     Alzheimer's disease; neuroprotective; nootropic.
KW
XX
     Homo sapiens.
OS
XX
PN
     US6022859-A.
XX
     08-FEB-2000.
PD
XX
                    97US-00970833.
     14-NOV-1997;
PF
XX
                    96US-0030840P.
     15-NOV-1996;
PR
XX
     (WISC ) WISCONSIN ALUMNI RES FOUND.
PΑ
XX
                 Kiessling LL;
PI
     Murphy RM,
XX
     WPI; 2000-160387/14.
DR
XX
     Beta-amyloid inhibitor useful for treating Alzheimer's disease.
PT
XX
PS
     Example; Col 7; 15pp; English.
XX
CC
     The present invention describes a beta-amyloid inhibitor peptide. Beta-
     amyloid inhibitors have neuroprotective and nootropic properties. The
CC
     inhibitor peptides are useful for the treatment of Alzheimer's disease.
CC
     The present sequence represents a beta-amyloid recognition peptide used
CC
     in the exemplification of present invention
CC
XX
SQ
     Sequence 10 AA;
                          85.4%; Score 35; DB 3; Length 10;
  Query Match
                          100.0%; Pred. No. 1.4;
  Best Local Similarity
                                                                  0; Gaps
                                                                              0;
                               0; Mismatches
                                                   0; Indels
             7; Conservative
            1 LVFFAED 7
Qy
              Db
            2 LVFFAED 8
RESULT 15
AAB46229
     AAB46229 standard; peptide; 10 AA.
ID
XX
AC
     AAB46229;
XX
DT
     04-APR-2001 (first entry)
XX
     Human APP derived immunogenic peptide #25.
DE
XX
     Amyloid deposit; APP; Abeta; brain; human; clearing response; nootropic;
KW
     Fc receptor mediated phagocytosis; immunogenic response; neuroprotective;
KW
     amyloid precursor protein; Alzheimer's disease.
ΚW
```

```
XX
OS
    Homo sapiens.
XX
    WO200072880-A2.
PN
XX
     07-DEC-2000.
PD
XX
     26-MAY-2000; 2000WO-US014810.
PF
XX
     28-MAY-1999;
                   99US-00322289.
PR
XX
     (NEUR-) NEURALAB LTD.
PA
XX
     Schenk DB, Bard F, Vasquez NJ, Yednock T;
PΙ
XX
    WPI; 2001-032104/04.
DR
XX
     Preventing or treating a disease associated with amyloid deposits,
PT
     especially Alzheimer's disease, comprises administering amyloid specific
PT
     antibody.
PT
XX
     Disclosure; Fig 19; 143pp; English.
PS
XX
CC
     This invention describes a novel method of preventing or treating a
     disease associated with amyloid deposits of amyloid precursor protein
CC
     (APP) Abeta fragments in the brain of a patient, which comprises
CC
     administering to the patient: (a) an antibody that binds to Abeta, the
CC
     antibody binds to an amyloid deposit and induces a clearing response (Fc
CC
     receptor mediated phagocytosis) against it (b) a polypeptide containing
CC
     an N-terminal segment of at least residues 1-5 of Abeta; or (c) an agent
CC
     that induces an immunogenic response against residues 1-3 to 7-11 of
CC
     Abeta. The products of the invention have nootropic and neuroprotective
CC
     activity. The method is also useful for monitoring a course of treatment
CC
     being administered to a patient e.g. active and passive immunization. The
CC
     methods are useful for prophylactic and therapeutic treatment of
CC
CC
     Alzheimer's disease
XX
SQ
     Sequence 10 AA;
                          85.4%; Score 35; DB 4; Length 10;
  Query Match
                          100.0%; Pred. No. 1.4;
  Best Local Similarity
                                                                 0; Gaps
                                                                             0;
                              0; Mismatches
                                                   0; Indels
            7; Conservative
  Matches
            1 LVFFAED 7
Qу
              1 LVFFAED 7
Db
RESULT 16
AAB46226
ID
     AAB46226 standard; peptide; 10 AA.
XX
     AAB46226;
AC
XX
DΤ
     04-APR-2001 (first entry)
XX
     Human APP derived immunogenic peptide #22.
DE
```

```
XX
     Amyloid deposit; APP; Abeta; brain; human; clearing response; nootropic;
KW
     Fc receptor mediated phagocytosis; immunogenic response; neuroprotective;
KW
     amyloid precursor protein; Alzheimer's disease.
KW
XX
OS
     Homo sapiens.
XX
     WO200072880-A2.
PN
XX
     07-DEC-2000.
PD
XX
     26-MAY-2000; 2000WO-US014810.
PF
XX
                    99US-00322289.
PR
     28-MAY-1999;
XX
     (NEUR-) NEURALAB LTD.
PA
XX
     Schenk DB, Bard F, Vasquez NJ, Yednock T;
PΙ
XX
DR
     WPI; 2001-032104/04.
XX
     Preventing or treating a disease associated with amyloid deposits,
PT
     especially Alzheimer's disease, comprises administering amyloid specific
PT
PT
     antibody.
XX
PS
     Disclosure; Fig 19; 143pp; English.
XX
     This invention describes a novel method of preventing or treating a
CC
CC
     disease associated with amyloid deposits of amyloid precursor protein
     (APP) Abeta fragments in the brain of a patient, which comprises
CC
     administering to the patient: (a) an antibody that binds to Abeta, the
CC
     antibody binds to an amyloid deposit and induces a clearing response (Fc
CC
     receptor mediated phagocytosis) against it (b) a polypeptide containing
CC
     an N-terminal segment of at least residues 1-5 of Abeta; or (c) an agent
CC
     that induces an immunogenic response against residues 1-3 to 7-11 of
CC
     Abeta. The products of the invention have nootropic and neuroprotective
CC
     activity. The method is also useful for monitoring a course of treatment
CC
     being administered to a patient e.g. active and passive immunization. The
CC
     methods are useful for prophylactic and therapeutic treatment of
CC
CC
     Alzheimer's disease
XX
SO
     Sequence 10 AA;
                          85.4%; Score 35; DB 4; Length 10;
  Query Match
                          100.0%; Pred. No. 1.4;
  Best Local Similarity
                                                                              0;
                                                   0; Indels
                                                                  0;
                                                                      Gaps
             7: Conservative
                                0; Mismatches
Qу
            1 LVFFAED 7
              Db
            4 LVFFAED 10
RESULT 17
AAB46228
     AAB46228 standard; peptide; 10 AA.
ID
XX
AC
     AAB46228;
```

```
XX
     04-APR-2001 (first entry)
DT
XX
     Human APP derived immunogenic peptide #24.
DE
XX
     Amyloid deposit; APP; Abeta; brain; human; clearing response; nootropic;
KW
     Fc receptor mediated phagocytosis; immunogenic response; neuroprotective;
KW
     amyloid precursor protein; Alzheimer's disease.
KW
XX
OS
     Homo sapiens.
XX
PN
     WO200072880-A2.
XX
     07-DEC-2000.
PD
XX
     26-MAY-2000; 2000WO-US014810.
PF
XX
     28-MAY-1999;
                    99US-00322289.
PR
XX
PA
     (NEUR-) NEURALAB LTD.
XX
PI
     Schenk DB, Bard F, Vasquez NJ, Yednock T;
XX
DR
     WPI; 2001-032104/04.
XX
     Preventing or treating a disease associated with amyloid deposits,
PT
     especially Alzheimer's disease, comprises administering amyloid specific
PT
PT
     antibody.
XX
PS
     Disclosure; Fig 19; 143pp; English.
XX
     This invention describes a novel method of preventing or treating a
CC
     disease associated with amyloid deposits of amyloid precursor protein
CC
     (APP) Abeta fragments in the brain of a patient, which comprises
CC
     administering to the patient: (a) an antibody that binds to Abeta, the
CC
     antibody binds to an amyloid deposit and induces a clearing response (Fc
CC
     receptor mediated phagocytosis) against it (b) a polypeptide containing
CC
     an N-terminal segment of at least residues 1-5 of Abeta; or (c) an agent
CC
     that induces an immunogenic response against residues 1-3 to 7-11 of
CC
     Abeta. The products of the invention have nootropic and neuroprotective
CC
     activity. The method is also useful for monitoring a course of treatment
CC
     being administered to a patient e.g. active and passive immunization. The
CC
     methods are useful for prophylactic and therapeutic treatment of
CC
     Alzheimer's disease
CC
XX
SO
     Sequence 10 AA;
                          85.4%; Score 35; DB 4; Length 10;
  Query Match
  Best Local Similarity
                          100.0%; Pred. No. 1.4;
                                                                     Gaps
                                                                              0;
             7: Conservative
                                0; Mismatches
                                                   0; Indels
                                                                  0;
Qу
            1 LVFFAED 7
              Db
            2 LVFFAED 8
```

```
AAB46227 standard; peptide; 10 AA.
TD
XX
AC
     AAB46227;
XX
DT
     04-APR-2001 (first entry)
XX
     Human APP derived immunogenic peptide #23.
DΕ
XX
     Amyloid deposit; APP; Abeta; brain; human; clearing response; nootropic;
KW
     Fc receptor mediated phagocytosis; immunogenic response; neuroprotective;
ΚW
     amyloid precursor protein; Alzheimer's disease.
KW
XX
OS
     Homo sapiens.
XX
     WO200072880-A2.
PN
XX
     07-DEC-2000.
PD
XX
     26-MAY-2000; 2000WO-US014810.
PF
XX
PR
     28-MAY-1999;
                    99US-00322289.
XX
PA
     (NEUR-) NEURALAB LTD.
XX
     Schenk DB, Bard F, Vasquez NJ, Yednock T;
PΙ
XX
     WPI; 2001-032104/04.
DR
XX
     Preventing or treating a disease associated with amyloid deposits,
PT
     especially Alzheimer's disease, comprises administering amyloid specific
PT
PT
     antibody.
XX
     Disclosure; Fig 19; 143pp; English.
PS
XX
     This invention describes a novel method of preventing or treating a
CC
     disease associated with amyloid deposits of amyloid precursor protein
CC
     (APP) Abeta fragments in the brain of a patient, which comprises
CC
     administering to the patient: (a) an antibody that binds to Abeta, the
CC
     antibody binds to an amyloid deposit and induces a clearing response (Fc
CC
     receptor mediated phagocytosis) against it (b) a polypeptide containing
CC
     an N-terminal segment of at least residues 1-5 of Abeta; or (c) an agent
CC
     that induces an immunogenic response against residues 1-3 to 7-11 of
CC
     Abeta. The products of the invention have nootropic and neuroprotective
CC
     activity. The method is also useful for monitoring a course of treatment
CC
     being administered to a patient e.g. active and passive immunization. The
CC
     methods are useful for prophylactic and therapeutic treatment of
CC
     Alzheimer's disease
CC
XX
SQ
     Sequence 10 AA;
                           85.4%; Score 35; DB 4; Length 10;
  Query Match
                          100.0%; Pred. No. 1.4;
  Best Local Similarity
                                 0; Mismatches
                                                    0; Indels
                                                                  0; Gaps
                                                                              0;
  Matches
             7; Conservative
            1 LVFFAED 7
Qy
```

111111

CC

```
RESULT 19
AAW32560
     AAW32560 standard; peptide; 11 AA.
ID
XX
AC
     AAW32560;
XX
DT
     21-JAN-1998
                 (first entry)
XX
     Anti-amyloid peptide Abeta inhibiting abnormal protein folding.
DE
XX
     Anti-amyloid peptide; iAbeta; abnormal protein folding inhibitor;
KW
     Alzheimer's disease; dementia; Down's syndrome; amyloidosis disorder;
KW
     human prion disease; Kuru; Creutzfeldt-Jakob disease;
KW
     Gerstmann-Straussler-Scheinker Syndrome; animal prion disease;
KW
     prion associated human neurodegenerative disease; scrapie;
KW
     spongiform encephalopathy; transmissible mink encephalopathy;
ΚW
     chronic wasting disease; mule; deer; elk; human.
KW
XX
OS
     Homo sapiens.
OS
     Synthetic.
XX
PN
     WO9639834-A1.
XX
PD
     19-DEC-1996.
XX
     06-JUN-1996;
                    96WO-US010220.
PF
XX
     07-JUN-1995;
                    95US-00478326.
PR
     10-APR-1996;
                    96US-00630645.
PR
XX
PΑ
     (UYNY ) UNIV NEW YORK STATE.
XX
ΡI
     Soto-Jara C, Baumann MH, Frangione B;
XX
DR
     WPI; 1997-051637/05.
XX
     New inhibitors of fibrillogenesis proteins or peptides - used for
PT
     preventing, treating or detecting amyloidosis disorders such as
PT
PΤ
     Alzheimer's disease.
XX
     Example 1; Fig 9; 63pp; English.
PS
XX
     A method has been developed for the prevention or treatment of a disorder
CC
     or disease associated with the formation of amyloid or amyloid-like
CC
     deposits, involving the abnormal folding of a protein or peptide. The
CC
     method involves administering an inhibitory peptide which prevents the
CC
     abnormal folding or which dissolves existing amyloid or amyloid-like
CC
     deposits, where the peptide comprises a sequence of 3-15 amino acid
CC
     residues and has a hydrophobic cluster of at least 3 amino acids, where
CC
     at least one of the 3 amino acids is a beta-sheet blocking amino acid
CC
     residue selected from Pro, Gly, Asn and His. The present sequence
CC
     represents an anti-amyloid peptide, Abeta, which inhibits abnormal
CC
     protein folding. The inhibitory peptide is capable of associating with a
CC
```

structural determinant on the protein or peptide to structurally block

```
and inhibit the abnormal folding into amyloid or amyloid-like deposits.
     The method can be used for preventing, treating or detecting e.g.
CC
     Alzheimer's dementia or disease, Down's syndrome, other amyloidosis
CC
     disorders, human prion diseases such as Kuru, Creutzfeldt-Jakob disease,
CC
     Gerstmann-Straussler-Scheinker Syndrome, prion associated human
CC
     neurodegenerative diseases or animal prion diseases such as scrapie,
CC
     spongiform encephalopathy, transmissible mink encephalopathy and chronic
CC
     wasting disease of mule deer and elk
CC
XX
SQ
     Sequence 11 AA;
                          85.4%; Score 35; DB 2; Length 11;
  Query Match
                          100.0%; Pred. No. 1.6;
  Best Local Similarity
                                                                             0;
                               0; Mismatches
                                                   0; Indels
                                                                 0; Gaps
 Matches
            7; Conservative
            1 LVFFAED 7
Qу
              3 LVFFAED 9
Db
RESULT 20
AAM52586
     AAM52586 standard; peptide; 11 AA.
ID
XX
AC
     AAM52586;
XX
DT
     07-FEB-2002 (first entry)
XX
     Peptide #16 for illustrating method of anticipating protein interaction.
DE
XX
KW
     Protein interaction; biochemistry; molecular biology; drug development;
     agrochemical; bioengineering.
KW
XX
os
     Unidentified.
XX
ΡN
     W0200167299-A1.
XX
PD
     13-SEP-2001.
XX
PF
     09-MAR-2001; 2001WO-JP001846.
XX
PR
     10-MAR-2000; 2000JP-00072485.
XX
PΑ
     (DAUC ) DAIICHI PHARM CO LTD.
PA
     (FUIT ) FUJITSU LTD.
XX
PΙ
     Doi H, Suzuki A;
XX
DR
     WPI; 2001-570799/64.
XX
     Method for assaying a specific protein for assaying anticipated
PT
PT
     information.
XX
PS
     Example 14; Page 34; 64pp; Japanese.
XX
     The present invention relates to a method for anticipating interaction
CC
     between proteins. The method comprises (1) digesting protein A into
CC
```

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oligopeptides; (2) searching a protein sequence database for polypeptides
     (polypeptide C) containing these oligopeptide sequences or D their
CC
     homologues; (3) performing a local alignment of A and detected C or D;
CC
     and (4) using a value calculated from the amino acid or oligonucleotide
CC
     frequencies, anticipating that C or D is polypeptide B that interacts
CC
     with A. The method is useful for assaying anticipated information about
CC
     proteins in biochemical, molecular biology, drug development,
CC
     agrochemical and bioengineering areas. The present sequence was used to
CC
     illustrate the method
CC
XX
SQ
     Sequence 11 AA;
                          85.4%; Score 35; DB 4; Length 11;
  Query Match
                          100.0%; Pred. No. 1.6;
  Best Local Similarity
                                                   0; Indels
                                                                 0; Gaps
                                                                             0;
            7; Conservative 0; Mismatches
            1 LVFFAED 7
Qу
              111111
Db
            2 LVFFAED 8
RESULT 21
AAU99431
     AAU99431 standard; peptide; 11 AA.
XX
AC
    AAU99431;
XX
     07-OCT-2002 (first entry)
DT
XX
     Human amyloid beta-peptide (1ba6) helical segment.
DΕ
XX
     I-helical conformation; discordant helix; amyloid beta-peptide; I-helix;
KW
     theta-strand structure; amyloidogenic disorder; Abeta; amyloidosis;
KW
KW
     Alzheimer's disease; prion disease; scrapie; BSE;
     bovine spongiform encephalopathy; Creutzfeld-Jacob disease; CJD;
KW
     fibrillation; aggregation; nootropic; neuroprotective; PDB;
KW
KW
     protein databank code; 1ba6; human.
XX
OS
     Homo sapiens.
XX
PN
     WO200241002-A2.
XX
PD
     23-MAY-2002.
XX
PF
     20-NOV-2001; 2001WO-GB005117.
XX
PR
     20-NOV-2000; 2000US-0253695P.
PR
     06-DEC-2000; 2000US-0251662P.
XX
PΑ
     (ALPH-) ALPHABETA AB.
PΑ
     (WHIT/) WHITE M P.
XX
PI
     White MP, Johansson J;
XX
DR
     WPI; 2002-519389/55.
XX
     Identifying compounds that stabilize I-helix of discordant helix in
PT
```

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polypeptide, by measuring amount of I-helix in sample containing
PT
     discordant helix-containing polypeptide in presence and absence of
PT
PT
     compound.
XX
     Example 1; Fig 2A; 55pp; English.
PS
XX
     The present invention relates to a method of identifying a compound that
CC
     stabilises an I-helical conformation of a discordant helix in a
CC
     polypeptide, particularly amyloid beta-peptide (Abeta). The method
CC
     comprises providing a test sample comprising a polypeptide that contains
CC
     a discordant helix in the form of an I-helix, contacting the test sample
CC
     with a test compound and determining the rate of decrease in the amount
CC
     of I-helix or the amount of I-helix present in the test sample. The
CC
     method is useful for identifying a compound that stabilises an I-helical
CC
     conformation of a discordant helix in a polypeptide. Such compounds are
CC
     useful for decreasing the rate of formation of theta-strand structures
CC
     between at least two discordant helix-containing polypeptides, and for
CC
     treating amyloidogenic disorders such as amyloidosis in Alzheimer's
CC
     disease, and prion diseases (e.g. scrapie, bovine spongiform
CC
     encephalopathy (BSE), Creutzfeld-Jacob disease (CJD)). AAU99426-AAU99446
CC
     represent >9-residue discordant helical segments from various proteins
CC
XX
SQ
     Sequence 11 AA;
                          85.4%; Score 35; DB 5; Length 11;
  Query Match
  Best Local Similarity
                          100.0%; Pred. No. 1.6;
                                                                              0;
             7; Conservative
                                0; Mismatches
                                                   0; Indels
                                                                  0; Gaps
  Matches
            1 LVFFAED 7
Qу
              1111111
            3 LVFFAED 9
Db
RESULT 22
AAE29504
     AAE29504 standard; peptide; 11 AA.
ID
XX
AC
     AAE29504;
XX
DT
     27-JAN-2003 (first entry)
XX
     Amyloid beta-protein related peptide #1.
DE
XX
     Metallopeptide; nootropic; amyloid beta-protein; Alzheimer's disease; AD;
KW
     Prion's disease; oxytocin; angiotensin; vasopressin; neuroprotective;
KW
     therapy; amyloid beta-protein related peptide.
KW
XX
OS
     Unidentified.
XX
PN
     WO200264734-A2.
XX
PD
     22-AUG-2002.
XX
     19-DEC-2001; 2001WO-US050075.
PF
XX
PR
     19-DEC-2000; 2000US-0256842P.
PR
     11-JUL-2001; 2001US-0304835P.
```

```
PR
     04-OCT-2001; 2001US-0327835P.
XX
     (PALA-) PALATIN TECHNOLOGIES INC.
PA
XX
     Sharma SD,
                Shi Y;
PΙ
XX
     WPI; 2002-740699/80.
DR
XX
     Determining secondary structure binding to desired targets within parent
PT
     polypeptides that bind to targets, by constructing and complexing
PT
     peptides to metal ions to form metallopeptides and screening the
PT
PT
     metallopeptides.
XX
     Claim 194; Page 98; 165pp; English.
PS
XX
     The invention relates to a method for identification and determination of
CC
     target-specific folding sites in peptides and proteins. The invention
CC
     also relates to a method for determining a secondary structure binding to
CC
     desired targets within parent polypeptides that bind to targets, by
CC
     constructing and complexing peptides to metal ions to form
CC
     metallopeptides and screening the metallopeptides. The method is useful
CC
     for determining secondary structure binding to desired target within
CC
     parent polypeptide with primary structure that binds to the target, where
CC
     the target of interest is a receptor, antibody, toxin, enzyme, hormone,
CC
     nucleic acid, intracellular protein domain of biological relevance or
CC
     extracellular protein domain of biological relevance. A library of
CC
     amyloid beta-protein related peptides is useful for the treatment of
CC
     Alzheimer's disease (AD). A library of peptides targetting vasopressin,
CC
     oxytocin or angiotensin receptor is useful for treating Prion's disease.
CC
     The present sequence is an amyloid beta-protein related peptide
CC
XX
     Sequence 11 AA;
SQ
  Query Match
                          85.4%; Score 35; DB 5;
                                                   Length 11;
                          100.0%; Pred. No. 1.6;
  Best Local Similarity
                                0; Mismatches
                                                   0; Indels
                                                                 0; Gaps
  Matches
             7; Conservative
            1 LVFFAED 7
Qy
              111111
Db
            4 LVFFAED 10
RESULT 23
ABU79013
     ABU79013 standard; peptide; 11 AA.
XX
AC
     ABU79013;
XX
DT
     17-JUN-2003 (first entry)
XX
DΕ
     Amyloidogenic Amyloid A peptide #3.
XX
     Amyloid formation; amyloid-like deposit; Alzheimer's disease;
KW
     pathological beta-sheet-rich conformation; Down's syndrome;
KW
     amyloidosis disorder; human prion disease; kuru; CJD;
KW
     Creutzfeldt-Jakob disease; Gerstmann-Straussler-Scheinker syndrome; GSS;
KW
     prion associated human neurodegenerative disease; animal prion disease;
KW
```

```
scrapie; spongiform encephalopathy; transmissible mink encephalopathy;
KW
     chronic wasting disease.
KW
XX
     Homo sapiens.
OS
XX
PN
    US6462171-B1.
XX
PD
     08-OCT-2002.
XX
                    96US-00766596.
PF
     12-DEC-1996;
XX
                    95US-00478326.
PR
     07-JUN-1995;
                    96US-00630645.
PR
     10-APR-1996;
XX
     (UYNY ) UNIV NEW YORK STATE.
PA
XX
     Soto-Jara C, Baumann MH, Frangione B;
PI
XX
DR
     WPI; 2003-379012/36.
XX
     Novel inhibitory peptides which inhibit and structurally block abnormal
PT
     folding of protein into amyloid or amyloid-like deposit and into
PT
     pathological beta-sheet rich conformation, useful for treating
PT
     Alzheimer's disease.
PT
XX
PS
     Disclosure; Fig 9; 51pp; English.
XX
     The invention describes an isolated inhibitory peptide (I) which
CC
     interacts with a hydrophobic beta-sheet forming cluster of amino acid
CC
     residues on a protein or peptide for amyloid or amyloid-like deposit
CC
     formation, and inhibits or structurally blocks the abnormal folding of
CC
     proteins and peptides into amyloid or amyloid-like deposits and into
CC
     pathological beta-sheet-rich conformation. (I) is useful for disorders or
CC
     diseases associated with abnormal protein folding into amyloid or amyloid
CC
     -like deposits or into pathological beta-sheet-rich precursors of such
CC
     deposits, such as Alzheimer's disease, Down's syndrome, other amyloidosis
CC
     disorders, human prion diseases, such as kuru, Creutzfeldt-Jakob disease
CC
     (CJD), Gerstmann-Straussler-Scheinker syndrome (GSS), prion associated
CC
     human neurodegenerative diseases as well as animal prion diseases such as
CC
     scrapie, spongiform encephalopathy, transmissible mink encephalopathy and
CC
     chronic wasting disease of mule deer and elk. (I) is also useful for
CC
     detecting and diagnosing the presence or absence of amyloid or amyloid-
CC
     like deposits in vivo and its precursors. This is the amino acid sequence
CC
     of peptide associated with the inhibition of amyloid or amyloid like
CC
CC
     deposits
XX
SQ
     Sequence 11 AA;
                          85.4%; Score 35; DB 6; Length 11;
  Query Match
  Best Local Similarity
                          100.0%; Pred. No. 1.6;
                                                                              0;
             7; Conservative
                                                   0; Indels
                                                                 0; Gaps
  Matches
                               0; Mismatches
Qу
            1 LVFFAED 7
              3 LVFFAED 9
Db
```

```
RESULT 24
ABW00147
     ABW00147 standard; peptide; 11 AA.
ID
XX
     ABW00147;
AC
XX
     15-JAN-2004 (first entry)
DT
XX
     Amyloid-beta (Abeta) peptide.
DE
XX
     Amyloid-like fibril deposit; prion related encephalopathy; gene therapy;
KW
     Alzheimer's disease; amyloid-beta; Abeta.
KW
XX
OS
     Unidentified.
XX
     US2003087407-A1.
PN
XX
     08-MAY-2003.
PD
XX
     06-SEP-2002; 2002US-00235483.
ΡF
XX
PR
     07-JUN-1995;
                    95US-00478326.
                    96US-00630645.
PR
     10-APR-1996;
                    96US-00766596.
PR
     12-DEC-1996;
XX
PA
     (UYNY ) UNIV NEW YORK STATE.
XX
     Soto-Jara C, Baumann MH, Frangione B;
ΡI
XX
DR
     WPI; 2003-616149/58.
XX
     New inhibitory peptide, useful for preparing a composition for
PT
     diagnosing, preventing or treating disorders associated with amyloid-like
PT
     fibril deposits, e.g. Alzheimer's disease, or prion related
PT
PT
     encephalopathies.
XX
PS
     Disclosure; Fig 9; 52pp; English.
XX
     The invention relates to inhibitory peptide comprising a portion of at
CC
     least three amino acid residues and a sequence predicted not to adopt a
CC
     beta-sheet structure that associates with a hydrophobic beta-sheet
CC
     cluster on a protein or peptide involved in the abnormal folding into a
CC
     beta-sheet structure, to structurally block the abnormal folding of the
CC
     protein or peptide. The inhibitory peptide is useful for preparing a
CC
     composition for preventing, treating or detecting disorders or diseases
CC
     associated with amyloid-like fibril deposits e.g. Alzheimer's disease and
CC
     prion related encephalopathies. The invention is also useful in gene
CC
     therapy. The present sequence is amyloid-beta (Abeta) peptide. This
CC
     peptide is used in the invention
CC
XX
SQ
     Sequence 11 AA;
                          85.4%; Score 35; DB 7; Length 11;
  Query Match
  Best Local Similarity 100.0%; Pred. No. 1.6;
                                                   0; Indels
                                                                 0; Gaps
                                                                              0;
            7; Conservative
                               0; Mismatches
  Matches
```

```
RESULT 25
AAR60372
     AAR60372 standard; peptide; 12 AA.
XX
     AAR60372;
AC
XX
     25-MAR-2003
                  (revised)
DT
DT
     15-MAR-1995
                  (first entry)
XX
     Beta-amyloid (17-28).
DΕ
XX
     Amyloid precursor protein; APP; Alzheimer's disease; beta-amyloid;
KW
     anti-beta-amyloid antibody; diagnosis.
KW
XX
OS
     Homo sapiens.
XX
PN
     WO9417197-A1.
XX
PD
     04-AUG-1994.
XX
                    94WO-JP000089.
ΡF
     24-JAN-1994;
XX
                    93JP-00010132.
PR
     25-JAN-1993;
                    93JP-00019035.
PR
     05-FEB-1993;
     16-NOV-1993;
                    93JP-00286985.
PR
     28-DEC-1993;
                    93JP-00334773.
PR
XX
     (TAKE ) TAKEDA CHEM IND LTD.
PA
XX
PΙ
     Suzuki N, Odaka A, Kitada C;
XX
DR
     WPI; 1994-264110/32.
XX
     Antibodies recognising specific parts of beta-amyloid - can be used for
PT
     diagnosis of diseases implicating beta-amyloid, such as Alzheimer's
PT
     disease.
PT
XX
PS
     Disclosure; Page 85; 116pp; Japanese.
XX
     Antibodies which recognise specific subfragments of the beta-amyloid
CC
     protein are claimed. Specifically, the antibodies (which are pref.
CC
     monoclonal) recognise residues 1-16 and/or 1-28 from the N-terminal
CC
     portion of beta-amyloid or they recognise residues 25-35 or 35-43 from
CC
     the C-terminal portion. The antibodies are useful for assaying beta-
CC
     amyloid and its derivatives for diagnosis of Alzheimer's disease.
CC
CC
     (Updated on 25-MAR-2003 to correct PN field.)
XX
SQ
     Sequence 12 AA;
                           85.4%; Score 35; DB 2; Length 12;
  Query Match
                           100.0%; Pred. No. 1.7;
  Best Local Similarity
                                  0; Mismatches
                                                    0; Indels
                                                                   0; Gaps
             7; Conservative
```

0;

```
1 LVFFAED 7
Qy
              111111
            1 LVFFAED 7
Db
RESULT 26
AAB10957
     AAB10957 standard; protein; 12 AA.
XX
     AAB10957;
AC
XX
     07-FEB-2001 (first entry)
DT
XX
     Bovine ADAM 10 peptide substrate #2.
DE
XX
     APP; amyloid precursor protein; human; alpha-secretase; ADAM 10;
KW
     disintegrin-metalloprotease; protease; nootropic; neuroprotective;
KW
     gene therapy; Alzheimer's disease.
KW
XX
OS
     Bos taurus.
XX
PN
     DE19910108-A1.
XX
     21-SEP-2000.
PD
XX
PF
     08-MAR-1999;
                    99DE-01010108.
XX
     08-MAR-1999;
                    99DE-01010108.
PR
XX
PΑ
     (FAHR/) FAHRENHOLZ F.
XX
PΙ
     Fahrenholz F, Postina R;
XX
DR
     WPI; 2000-588391/56.
XX
     Recombinant cells, for identifying alpha-secretase active agents and
PT
     identifying risk factors associated with Alzheimer's disease, comprise
PT
     amyloid precursor protein and alpha-secretase.
PT
XX
     Disclosure; Page 6; 24pp; German.
PS
XX
     This invention describes a novel recombinant cell comprising recombinant
CC
     nucleic acids encoding a region of human amyloid precursor protein
CC
     containing an alpha-secretase cleavage site and a protease or a
CC
     heterologous RNA coding for a substrate protein and a protease. The
CC
     invention also describes a recombinant cell, characterized in that it
CC
     contains recombinant nucleic acids comprising either: (a) a gene for a
CC
     substrate protein (SP), which comprises a sequence region of 18 amino
CC
     acids of the human amyloid precursor protein (APP) or a homologous
CC
     protein, where the sequence region contains the alpha-secretase cleavage
CC
     site at a reference of 6 residues at the N-terminal and 12 residues at
CC
     the C-terminal; and (b) a gene for a protease protein (PP), that either
CC
     comprises a proteolytically active necessary sequence region or a
CC
     sequence region of the disintegrin metalloprotease ADAM 10 from a cow
CC
     (Bos taurus), from a human or other mammal or a mutant of this, which
CC
     shows the same enzymatic properties, where the genes are under the
```

control of heterologous promoters; or a heterologous RNA coding for a SP

CC

```
and a PP. The products of the invention have nootropic and
CC
    neuroprotective activity and can be used for gene therapy. The protease
CC
    proteins of the invention are useful for proteolytic cleavage of
CC
    substrate proteins, especially human amyloid precursor protein. Dominant
CC
    negative forms of bovine, human or other mammalian disintegrin-
CC
    metalloprotease ADAM 10 proteins and their coding sequences are useful
CC
     for suppressing the alpha-secretase activity of a cell. Nucleic acid
CC
     sequences encoding the proteases are useful for constructing vectors for
CC
     gene therapy. The proteins and recombinant cells are useful for
CC
     identifying secretases and pharmaceutical agents and to identify risk
CC
     factors associated with Alzheimer's disease
CC
XX
SO
     Sequence 12 AA;
                          85.4%; Score 35; DB 3; Length 12;
 Query Match
                          100.0%; Pred. No. 1.7;
  Best Local Similarity
                              0; Mismatches
                                                                 0; Gaps
                                                                             0;
                                                   0; Indels
            7; Conservative
            1 LVFFAED 7
Qy
              1 LVFFAED 7
RESULT 27
AAE35466
     AAE35466 standard; peptide; 12 AA.
XX
AC
     AAE35466;
XX
DT
     17-JUN-2003 (first entry)
XX
DΕ
     Abeta peptide #37.
XX
     All-D-amyloid-beta peptide; Alzheimer's disease; rheumatoid arthritis;
KW
     cerebral amyloid angiopathy; amyloid disease; ankylosing spondylitis;
KW
     psoriasis; Reiter's syndrome; Adult Still's disease; Bechet's syndrome;
KW
     Crohn's disease; infection; leprosy; tuberculosis; carcinoma; nootropic;
KW
     chronic pyelonephritis; osteomyelitis; Whipple's disease; vasotropic;
KW
     Hodgkin's lymphoma; neuroprotective; bronchiectasis; ophthalmological;
KW
     ulcer; antiinflammatory; cytostatic; uropathic; therapy.
KW
XX
OS
     Unidentified.
XX
                     Location/Qualifiers
FH
     Kev
     Misc-difference 1. .12
FT
                     /note= "D-form residues"
FT
XX
     WO200296937-A2.
PN
XX
PD
     05-DEC-2002.
XX
     29-MAY-2002; 2002WO-CA000763.
PF
XX
     29-MAY-2001; 2001US-00867847.
PR
XX
PΑ
     (NEUR-) NEUROCHEM INC.
XX
```

```
Gervais F, Hebert L, Chalifour RJ, Kong X;
PΙ
XX
     WPI; 2003-201269/19.
DR
XX
     Prevention and/or treatment of an amyloid-related disease e.g.
PT
     Alzheimer's disease, comprises use of all-D-amyloid-beta peptides.
PT
XX
     Claim 1; Page 61; 44pp; English.
PS
XX
     The invention relates to a method for prevention and/or treatment of an
CC
     amyloid-related disease which comprises administration of an all-D -
CC
     amyloid-beta peptide. The method is used for preventing and/or treating
CC
     Alzheimer's and other amyloid related disease e.g. cerebral amyloid
CC
     angiopathy; for altering serum levels of amyloid-beta in a mammal and
CC
     favours the clearance of soluble amyloid-beta or fibril amyloid-beta from
CC
     the mammal; and reducing or inhibiting the formation of plaques. It is
CC
     also used for treating AA (reactive) amyloid diseases including
CC
     inflammatory diseases e.g. rheumatoid arthritis, juvenile chronic
CC
     arthritis, ankylosing spondylitis, psoriasis, psoriatic arthropathy,
CC
     Reiter's syndrome, Adult Still's disease, Bechet's syndrome and Crohn's
CC
     disease. AA deposits are also produced as a result of chronic microbial
CC
     infections (preferably leprosy, tuberculosis, bronchiectasis, decubitus
CC
     ulcers, chronic pyelonephritis, osteomyelitis and Whipple's disease).
CC
     Certain malignant neoplasms can also result in AA fibril amyloid deposits
CC
     including Hodgkin's lymphoma, renal carcinoma, carcinomas of gut, lung
CC
     and urogenital tract, basal cell carcinoma and hairy cell leukaemia. The
CC
     present sequence is an Abeta peptide used to illustrate the method of the
CC
CC
     invention
XX
SQ
     Sequence 12 AA;
                          85.4%; Score 35; DB 6; Length 12;
  Query Match
                          100.0%; Pred. No. 1.7;
  Best Local Similarity
                               0; Mismatches
                                                   0; Indels
                                                                 0; Gaps
                                                                             0;
  Matches
             7; Conservative
            1 LVFFAED 7
Qу
              5 LVFFAED 11
Db
RESULT 28
     AAE35465 standard; peptide; 13 AA.
XX
AC
     AAE35465;
XX
     17-JUN-2003 (first entry)
DТ
XX
DE
     Abeta peptide #36.
XX
     All-D-amyloid-beta peptide; Alzheimer's disease; rheumatoid arthritis;
KW
     cerebral amyloid angiopathy; amyloid disease; ankylosing spondylitis;
K₩
     psoriasis; Reiter's syndrome; Adult Still's disease; Bechet's syndrome;
ΚW
     Crohn's disease; infection; leprosy; tuberculosis; carcinoma; nootropic;
KW
     chronic pyelonephritis; osteomyelitis; Whipple's disease; vasotropic;
KW
     Hodgkin's lymphoma; neuroprotective; bronchiectasis; ophthalmological;
KW
     ulcer; antiinflammatory; cytostatic; uropathic; therapy.
KW
```

```
XX
     Unidentified.
OS
XX
                     Location/Qualifiers
FΗ
     Misc-difference 1. .6
FT
                     /note= "D-form residues"
FT
XX
PN
     WO200296937-A2.
XX
     05-DEC-2002.
PD
XX
     29-MAY-2002; 2002WO-CA000763.
PF
XX
     29-MAY-2001; 2001US-00867847.
PR
XX
     (NEUR-) NEUROCHEM INC.
PA
XX
     Gervais F, Hebert L, Chalifour RJ, Kong X;
PΙ
XX
DR
     WPI; 2003-201269/19.
XX
     Prevention and/or treatment of an amyloid-related disease e.g.
PT
     Alzheimer's disease, comprises use of all-D-amyloid-beta peptides.
PT
XX
     Claim 1; Page 61; 44pp; English.
PS
XX
     The invention relates to a method for prevention and/or treatment of an
CC
     amyloid-related disease which comprises administration of an all-D -
CC
     amyloid-beta peptide. The method is used for preventing and/or treating
CC
CC
     Alzheimer's and other amyloid related disease e.g. cerebral amyloid
     angiopathy; for altering serum levels of amyloid-beta in a mammal and
CC
     favours the clearance of soluble amyloid-beta or fibril amyloid-beta from
CC
     the mammal; and reducing or inhibiting the formation of plaques. It is
CC
CC
     also used for treating AA (reactive) amyloid diseases including
     inflammatory diseases e.g. rheumatoid arthritis, juvenile chronic
CC
     arthritis, ankylosing spondylitis, psoriasis, psoriatic arthropathy,
CC
     Reiter's syndrome, Adult Still's disease, Bechet's syndrome and Crohn's
CC
     disease. AA deposits are also produced as a result of chronic microbial
CC
     infections (preferably leprosy, tuberculosis, bronchiectasis, decubitus
CC
     ulcers, chronic pyelonephritis, osteomyelitis and Whipple's disease).
CC
     Certain malignant neoplasms can also result in AA fibril amyloid deposits
CC
     including Hodgkin's lymphoma, renal carcinoma, carcinomas of gut, lung
CC
     and urogenital tract, basal cell carcinoma and hairy cell leukaemia. The
CC
     present sequence is an Abeta peptide used to illustrate the method of the
CC
CC
     invention
XX
SQ
     Sequence 13 AA;
  Query Match
                          85.4%;
                                  Score 35; DB 6; Length 13;
  Best Local Similarity
                          100.0%; Pred. No. 1.9;
                                                                              0;
  Matches
             7; Conservative
                                0; Mismatches
                                                    0; Indels
Qy
            1 LVFFAED 7
              111111
Db
            2 LVFFAED 8
```

```
RESULT 29
AAE35467
     AAE35467 standard; peptide; 13 AA.
XX
AC
     AAE35467;
XX
     17-JUN-2003 (first entry)
DT
XX
     Abeta peptide #38.
DE
XX
     All-D-amyloid-beta peptide; Alzheimer's disease; rheumatoid arthritis;
KW
     cerebral amyloid angiopathy; amyloid disease; ankylosing spondylitis;
KW
     psoriasis; Reiter's syndrome; Adult Still's disease; Bechet's syndrome;
KW
     Crohn's disease; infection; leprosy; tuberculosis; carcinoma; nootropic;
KW
     chronic pyelonephritis; osteomyelitis; Whipple's disease; vasotropic;
KW
     Hodgkin's lymphoma; neuroprotective; bronchiectasis; ophthalmological;
     ulcer; antiinflammatory; cytostatic; uropathic; therapy.
KW
XX
OS
     Unidentified.
XX
FH
     Kev
                     Location/Qualifiers
FT
     Misc-difference 1. .13
                     /note= "D-form residues"
FT
XX
     WO200296937-A2.
PN
XX
PD
     05-DEC-2002.
XX
PF
     29-MAY-2002; 2002WO-CA000763.
XX
PR
     29-MAY-2001; 2001US-00867847.
XX
PA
     (NEUR-) NEUROCHEM INC.
XX
PΙ
     Gervais F, Hebert L, Chalifour RJ, Kong X;
XX
DR
     WPI; 2003-201269/19.
XX
     Prevention and/or treatment of an amyloid-related disease e.g.
PT
     Alzheimer's disease, comprises use of all-D-amyloid-beta peptides.
PT
XX
PS
     Claim 1; Page 61; 44pp; English.
XX
     The invention relates to a method for prevention and/or treatment of an
CC
     amyloid-related disease which comprises administration of an all-D -
CC
     amyloid-beta peptide. The method is used for preventing and/or treating
CC
     Alzheimer's and other amyloid related disease e.g. cerebral amyloid
CC
     angiopathy; for altering serum levels of amyloid-beta in a mammal and
CC
     favours the clearance of soluble amyloid-beta or fibril amyloid-beta from
CC
     the mammal; and reducing or inhibiting the formation of plaques. It is
CC
     also used for treating AA (reactive) amyloid diseases including
CC
     inflammatory diseases e.g. rheumatoid arthritis, juvenile chronic
CC
     arthritis, ankylosing spondylitis, psoriasis, psoriatic arthropathy,
CC
     Reiter's syndrome, Adult Still's disease, Bechet's syndrome and Crohn's
CC
     disease. AA deposits are also produced as a result of chronic microbial
CC
     infections (preferably leprosy, tuberculosis, bronchiectasis, decubitus
CC
     ulcers, chronic pyelonephritis, osteomyelitis and Whipple's disease).
CC
```

```
Certain malignant neoplasms can also result in AA fibril amyloid deposits
     including Hodgkin's lymphoma, renal carcinoma, carcinomas of gut, lung
CC
     and urogenital tract, basal cell carcinoma and hairy cell leukaemia. The
CC
     present sequence is an Abeta peptide used to illustrate the method of the
CC
CC
     invention
XX
     Sequence 13 AA;
SQ
                          85.4%; Score 35; DB 6;
                                                    Length 13;
  Query Match
                          100.0%; Pred. No. 1.9;
  Best Local Similarity
            7; Conservative 0; Mismatches
                                                   0; Indels
                                                                  0; Gaps
                                                                              0;
  Matches
            1 LVFFAED 7
Qγ
              11111
            2 LVFFAED 8
Db
RESULT 30
ADA37467
ID
     ADA37467 standard; peptide; 13 AA.
XX
AC
     ADA37467;
XX
     20-NOV-2003 (first entry)
DT
XX
DE
     Human amyloid precursor protein fragment.
XX
     ADAM; a disintegrin and metalloprotease; G-protein coupled receptor;
KW
     GPCR; beta-amyloid precursor protein; APP; alpha-secretase site;
KW
KW
     Alzheimer's disease.
XX
OS
     Homo sapiens.
XX
PN
     US2003108978-A1.
XX
PD
     12-JUN-2003.
XX
PF
     25-OCT-2002; 2002US-00281458.
XX
     25-OCT-2001; 2001US-0337641P.
PR
XX
PΑ
     (CIAM/) CIAMBRONE G J.
PA
     (GIBB/) GIBBONS I.
XX
PI
     Ciambrone GJ, Gibbons I;
XX
DR
     WPI; 2003-626205/59.
XX
     Assaying activity of an a disintegrin and metalloprotease in whole cell
PT
     system combining soluble substrate with whole cell system, and
РΤ
PT
     determining amount of product.
XX
     Disclosure; Page 9; 34pp; English.
PS
XX
     The invention relates to the activity of a disintegrin and
CC
     metalloprotease (ADAM) in a whole cell system assayed by selecting a
CC
     soluble substrate that is specifically cleavable by the ADAM, combining
CC
```

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the soluble substrate with the whole cell system under conditions that
CC
     allow processing of the substrate to a product by the ADAM and
CC
CC
     determining the amount of the product as an indication of the ADAM
     activity. Also included is a method of determining the effect of a G-
CC
     protein coupled receptor (GPCR) on the activity of an ADAM in a whole
CC
     cell system comprising selecting a ligand known to modulate activity of
CC
     the GPCR and a soluble substrate that is cleavable by the ADAM, preparing
CC
     two mixtures of the whole cell system and the soluble substrate, where
CC
     only one of the mixtures contains the ligand, incubating the mixtures
CC
     under conditions that allow processing of the substrate to a product by
CC
     the ADAM, if the ADAM is active, determining the amount of the product
CC
     formed in each mixture and comparing the amount of product formed in
CC
     separate mixtures to determine effect of the GPCR on the ADAM activity.
CC
     The method may be adapted to assay the effect of a compound on the
CC
     cleavage of the Beta-amyloid precursor protein (APP) at its alpha-
CC
     secretase site by ADAM 17 or ADAM 10. The invention is used for the
CC
     assaying for the activity of an ADAM in a whole cell system. The assay
CC
     may be used in the diagnosis of diseases associated with ADAM activities
CC
     e.q. Alzheimer's disease. The present sequence is the human APP peptide
CC
     fragment containing the alpha-secretase site.
CC
XX
SO
     Sequence 13 AA;
                          85.4%; Score 35; DB 6; Length 13;
  Query Match
  Best Local Similarity
                          100.0%; Pred. No. 1.9;
                                                                              0;
             7; Conservative
                                 0; Mismatches
                                                    0;
                                                        Indels
                                                                  0; Gaps
  Matches
            1 LVFFAED 7
Qу
              111111
Db
            7 LVFFAED 13
RESULT 31
AAE03423
     AAE03423 standard; peptide; 14 AA.
XX
AC
     AAE03423;
XX
DT
     06-AUG-2001 (first entry)
XX
     Peptide corresponding to residues 17-30 of human APP.
DE
XX
     Human; antisense; amyloid precursor protein; APP; amyloid beta protein;
KW
     AbetaP; Alzheimer's disease; cognitive ability; antisense therapy;
KW
KW
     nootropic; neuroprotective.
XX
OS
     Homo sapiens.
XX
PN
     WO200142266-A1.
XX
PD
     14-JUN-2001.
XX
     08-DEC-2000; 2000WO-US033383.
PF
XX
     09-DEC-1999;
                    99US-00458481.
PR
XX
PA
     (UYSL-) UNIV SAINT LOUIS.
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XX
PΙ
     Kumar VB;
XX
     WPI; 2001-381626/40.
DR
XX
     Novel antisense compounds for modulating expression of amyloid beta
PT
     protein in cells or tissues and for preventing, treating conditions
PT
     associated with expression of amyloid beta protein, e.g. Alzheimer's
PT
PT
     disease.
XX
     Disclosure; Page 14; 70pp; English.
PS
XX
     The present invention relates to an antisense compound comprising
CC
     nucleotides complementary to a nucleic acid sequence coding for amyloid
CC
     precursor protein (APP) and which inhibits the expression of amyloid beta
CC
     protein (Abeta) portion of APP coding sequence while permitting the
CC
     expression of at least a portion of APP polynucleotide 5' to the Abeta
CC
     portion of APP coding sequence. This antisense compound is useful for
CC
     modulating the expression of Abeta in cells or tissues, for preventing or
CC
     treating a disease or condition associated with expression of Abeta, in
CC
     particular Alzheimer's disease. The antisense compound is also useful for
CC
     improving cognitive ability in a mammal having a disease or condition
CC
     associated with the expression of Abeta. Antisense compounds are used in
CC
     antisense therapy. The present sequence is a peptide corresponding to
CC
CC
     amino acid residues 17-30 of the AbetaP portion of human APP
XX
SQ
     Sequence 14 AA;
                          85.4%; Score 35; DB 4;
                                                   Length 14;
  Query Match
                          100.0%; Pred. No. 2.1;
  Best Local Similarity
                                                    0; Indels
                                                                  0; Gaps
                                                                              0;
  Matches
             7; Conservative
                                0; Mismatches
            1 LVFFAED 7
Qу
              1111111
Db
            1 LVFFAED 7
RESULT 32
ADA89887
     ADA89887 standard; peptide; 14 AA.
ID
XX
AC
     ADA89887;
XX
DT
     20-NOV-2003 (first entry)
XX
     Beta-A4 second region peptide SEQ ID NO:2.
DE
XX
     antibody molecule; antibody; beta-A4 peptide; Abeta4; neuroprotective;
KW
     nootropic; antiparkinsonian; gene therapy; amyloidogenesis;
KW
     amyloid-plaque formation; beta-amyloid plaque; immunisation; dementia;
KW
     Alzheimer's disease; motor neuropathy; Down's syndrome;
KW
     Creutzfeldt Jacob disease; hereditary cerebral haemorrhage; amyloidosis;
KW
     Parkinson's disease; HIV-related dementia; amyotrophic lateral sclerosis;
KW
     neuronal disorder; aging.
KW
XX
OS
     Synthetic.
OS
     Homo sapiens.
```

XX WO2003070760-A2. PNXX 28-AUG-2003. PDXX 20-FEB-2003; 2003WO-EP001759. PFXX 20-FEB-2002; 2002EP-00003844. PRXX (HOFF) HOFFMANN LA ROCHE & CO AG F. PΑ (MORP-) MORPHOSYS AG. PA XX Bardroff M, Bohrmann B, Brockhaus M, Huber W, Kretzschmar T; PΙ Loehning C, Loetscher H, Nordstedt C, Rothe C; PIXX WPI; 2003-663848/62. DR XX New antibody molecule capable of specifically recognizing two regions of PTthe beta-A4 peptide, useful for diagnosing, preventing or treating PTdiseases associated with amyloidogenesis or amyloid-plaque formation PTPT(e.g. dementia). XX Claim 1; Page 99; 312pp; English. $_{\mathrm{PS}}$ XX CC The present invention describes an antibody molecule (I) capable of specifically recognising two regions of the beta-A4 peptide/Abeta4. The CC CC CC CC CC CC cell comprising the vector of (2); (4) preparing (I), comprising CCCC CC CC

first region comprises the amino acid sequence Ala-Glu-Phe-Arg-His-Asp-Ser-Gly-Tyr ADA89886 or its fragment, and the second region comprises the amino acid sequence Val-His-His-Gln-Lys-Leu-Val-Phe-Phe-Ala-Glu-Asp-Val-Gly ADA89887 or its fragment. Also described: (1) a nucleic acid molecule encoding (I); (2) a vector comprising the nucleic acid of (1); (3) a host culturing the host cell of (3) under conditions that allow synthesis of (I) and recovering (I) from the culture; (5) a composition comprising (I) or an antibody molecule produced by method (4); (6) a kit comprising (I), nucleic acid of (1), vector of (2) or host cell of (3); (7) optimising (I); (8) testing the resulting Fab optimisation library by panning against Abeta/Abeta4; (9) identifying optimised clones; (10) expressing of selected, optimised clones; (11) preparing a pharmaceutical composition, comprising optimisation of (I), and formulating the optimised antibody/antibody molecule with a carrier; and (12) a pharmaceutical composition prepared by method (8). (I) has neuroprotective, nootropic and antiparkinsonian activities, and can be used in gene therapy. The antibody molecule (I), nucleic acid molecule, vector or host is useful in preparing a pharmaceutical composition for the prevention and/or treatment of a disease associated with amyloidogenesis and/or amyloid-plaque formation. The antibody molecule may also be used in preparing a diagnostic composition for the detection of the disease mentioned above. The antibody is used for the disintegration of beta-amyloid plaques or for passive immunisation against beta-amyloid plaque formation. In particular, the disease is dementia, Alzheimer's disease, motor neuropathy, Down's syndrome, Creutzfeldt Jacob disease, hereditary cerebral haemorrhage with amyloidosis Dutch type, Parkinson's disease, HIV-related dementia, amyotrophic lateral sclerosis or neuronal disorders related to aging. The present sequence is used in the exemplification of the present invention.

CC XX

CC

```
SQ
     Sequence 14 AA;
                          85.4%; Score 35; DB 6; Length 14;
 Query Match
                         100.0%; Pred. No. 2.1;
  Best Local Similarity
                                                   0; Indels
                                                                 0; Gaps
                                                                             0;
            7; Conservative
                                0; Mismatches
            1 LVFFAED 7
Qу
             6 LVFFAED 12
Db
RESULT 33
AAW02334
     AAW02334 standard; peptide; 15 AA.
ID
XX
     AAW02334;
AC
XX
     06-MAY-1997 (first entry)
DT
XX
DE
     Beta-amyloid peptide residues 16-30.
XX
     Beta-amyloid; modulator; amyloid plaque; brain lesion; amyloidosis;
KW
     cerebral blood vessel; Alzheimer's disease; amyloidogenic protein;
KW
     familial amyloid polyneuropathy; familial amyloid cardiomyopathy;
KW
     isolated cardiac amyloidosis; systemic senile amyloidosis; insulinoma;
KW
     bovine spongiform encephalopathy; Creutzfeldt-Jakob disease; urticaria;
KW
     adult-onset diabetes; familial Mediterranean fever; therapy; deafness;
KW
     scrapie; familial amyloid nephropathy; hereditary cerebral haemorrhage.
KW
XX
OS
     Synthetic.
XX
     WO9628471-A1.
PN
XX
PD
     19-SEP-1996.
XX
PF
     14-MAR-1996;
                    96WO-US003492.
XX
PR
     14-MAR-1995;
                    95US-00404831.
PR
     07-JUN-1995;
                    95US-00475579.
PR
     27-OCT-1995;
                    95US-00548998.
XX
PA
     (PHAR-) PHARM PEPTIDES INC.
XX
     Findeis MA, Benjamin H, Garnick MB, Gefter ML, Hundal A;
PI
     Kasman L, Musso G, Signer ER, Wakefield J, Reed MJ, Molineaux S;
PI
     Kubasek W, Chin J, Lee J, Kelley M;
PΙ
XX
DR
     WPI; 1996-433762/43.
XX
PΤ
     Modulators of amyloid aggregation - comprising, e.g. amyloidogenic
     protein coupled (in)directly to at least 1 modifying gp., useful in
PT
     treatment of Alzheimer's disease.
PT
XX
PS
     Claim 29; Page 82; 106pp; English.
```

AAW02333-W02336 represent beta-amyloid peptide fragments that can be used

in the modulator compounds of the invention. Beta-amyloid peptide is a 4

XX

CC

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kilodalton peptide that is the major protein component of amyloid
CC
     plaques. Amyloid plaques are present both in the brain lesions, and in
CC
CC
     the walls of cerebral blood vessels in Alzheimer's disease patients. The
     amyloid modulators of the invention comprise an amyloidogenic protein or
CC
     peptide (see AAW02310-W02336) coupled directly or indirectly to at least
CC
     one modifying group. The modifying group is preferably a cyclic,
CC
     heterocyclic, or polycyclic group, such as declain, a cholanyl group, a
CC
     biotin containing group, or a fluorescein containing group. These
CC
     compounds then modulate the aggregation of these sequences to natural
CC
     amyloid proteins or peptides when contacted with the natural
CC
     amyloidogenic proteins or peptides. The modulator compounds can be used
CC
     in the treatment of disorders associated with amyloidosis, such as
CC
     familial amyloid polyneuropathy, familial amyloid cardiomyopathy,
CC
     isolated cardiac amyloidosis, systemic senile amyloidosis, scrapie,
CC
     bovine spongiform encephalopathy, Creutzfeldt-Jakob disease, adult-onset
CC
     diabetes, insulinoma, familial Mediterranean fever, familial amyloid
CC
     nephropathy with urticaria and deafness, hereditary cerebral haemorrhage
CC
     and other types of amyloidosis. The modulators are also useful for the
CC
     treatment of disorders associated with beta-amyloidosis, especially
CC
CC
     Alzheimer's disease
XX
SQ
     Sequence 15 AA;
                          85.4%; Score 35; DB 2; Length 15;
  Query Match
  Best Local Similarity
                          100.0%; Pred. No. 2.2;
            7; Conservative
                                 0; Mismatches
                                                   0;
                                                       Indels
                                                                     Gaps
                                                                              0;
  Matches
            1 LVFFAED 7
Qy
              1111111
Db
            2 LVFFAED 8
RESULT 34
AAW89358
     AAW89358 standard; peptide; 15 AA.
XX
AC
     AAW89358;
XX
DΤ
     02-MAR-1999 (first entry)
XX
DE
     Beta-amyloid peptide derivative A-beta-11-25.
XX
     Human; beta-amyloid peptide; Alzheimer's disease; amyloidogenic protein;
KW
     aggregation; neurotoxicity; amyloidosis; Down's syndrome; cardiomyopathy;
KW
     familial amyloid polyneuropathy; bovine spongiform encephalopathy;
KW
KW
     Creutzfeldt-Jakob disease; bAP.
XX
OS
     Homo sapiens.
os
     Synthetic.
XX
PN
     US5854204-A.
XX
PD
     29-DEC-1998.
XX
PF
     14-MAR-1996;
                    96US-00612785.
XX
PR
     14-MAR-1995;
                    95US-00404831.
```

```
PR
     07-JUN-1995;
                    95US-00475579.
     27-OCT-1995;
                   95US-00548998.
PR
XX
     (PRAE-) PRAECIS PHARM INC.
PA
XX
    Hundal A, Gefter ML, Kasman L, Musso G, Molineaux S, Benjamin H;
PΙ
     Findeis MA, Chin J, Lee J, Kelley M, Reed M, Wakefield J;
PΙ
     Garnick MB, Kubasek W, Signer ER;
PI
XX
    WPI; 1999-094964/08.
DR
XX
    New peptide(s) derived from beta-amyloid peptide that inhibit amyloid
PT
     aggregation - and neurotoxicity, specifically for treatment and
PT
    prevention of Alzheimer's disease.
PT
XX
     Claim 6; Col 81-82; 52pp; English.
PS
XX
     The present invention describes beta-amyloid peptide (bAP) derivatives.
CC
     The bAP derivatives inhibit aggregation of amyloidogenic proteins and
CC
     peptides, specifically bAP, and their neurotoxicity, so are useful for
CC
     treating and preventing any disease involving amyloidosis, specifically
CC
    Alzheimer's disease but also Down's syndrome, familial amyloid
CC
    polyneuropathy or cardiomyopathy, bovine spongiform encephalopathy and
CC
     Creutzfeldt-Jakob disease. The bAP derivatives are also used to diagnose
CC
CC
     these diseases, in vitro or in vivo, by detecting binding of bAP to
CC
     labelled bAP derivatives. Some bAP derivatives inhibit bAP aggregation
CC
     even when bAP is present in molar excess. The present sequence represents
CC
     a bAP derivative
XX
SQ
     Sequence 15 AA;
                          85.4%; Score 35; DB 2; Length 15;
  Query Match
  Best Local Similarity
                          100.0%; Pred. No. 2.2;
                                                                             0;
  Matches
            7; Conservative 0; Mismatches
                                                   0; Indels
                                                                 0; Gaps
            1 LVFFAED 7
Qу
              111111
Db ·
            7 LVFFAED 13
RESULT 35
     AAW89354 standard; peptide; 15 AA.
XX
AC
     AAW89354;
XX
DΤ
     02-MAR-1999 (first entry)
XX
DE
     Beta-amyloid peptide derivative A-beta-16-30.
XX
     Human; beta-amyloid peptide; Alzheimer's disease; amyloidogenic protein;
KW
     aggregation; neurotoxicity; amyloidosis; Down's syndrome; cardiomyopathy;
KW
     familial amyloid polyneuropathy; bovine spongiform encephalopathy;
KW
KW
     Creutzfeldt-Jakob disease; bAP.
XX
OS
     Homo sapiens.
OS
     Synthetic.
```

```
XX
PN
    US5854204-A.
XX
    29-DEC-1998.
PD
XX
                    96US-00612785.
PF
    14-MAR-1996;
XX
                    95US-00404831.
PR
    14-MAR-1995;
                    95US-00475579.
PR
     07-JUN-1995;
    27-OCT-1995;
                    95US-00548998.
PR
XX
     (PRAE-) PRAECIS PHARM INC.
PΑ
XX
    Hundal A, Gefter ML, Kasman L, Musso G, Molineaux S, Benjamin H;
PI
    Findeis MA, Chin J, Lee J, Kelley M, Reed M, Wakefield J;
PΙ
    Garnick MB, Kubasek W, Signer ER;
PΙ
XX
    WPI; 1999-094964/08.
DR
XX
    New peptide(s) derived from beta-amyloid peptide that inhibit amyloid
PT
     aggregation - and neurotoxicity, specifically for treatment and
PT
PT
    prevention of Alzheimer's disease.
XX
PS
    Claim 2; Col 71-72; 52pp; English.
XX
    The present invention describes beta-amyloid peptide (bAP) derivatives.
CC
    The bAP derivatives inhibit aggregation of amyloidogenic proteins and
CC
     peptides, specifically bAP, and their neurotoxicity, so are useful for
CC
     treating and preventing any disease involving amyloidosis, specifically
CC
     Alzheimer's disease but also Down's syndrome, familial amyloid
CC
    polyneuropathy or cardiomyopathy, bovine spongiform encephalopathy and
CC
    Creutzfeldt-Jakob disease. The bAP derivatives are also used to diagnose
CC
     these diseases, in vitro or in vivo, by detecting binding of bAP to
CC
     labelled bAP derivatives. Some bAP derivatives inhibit bAP aggregation
CC
     even when bAP is present in molar excess. The present sequence represents
CC
CC
     a bAP derivative
XX
SQ
     Sequence 15 AA;
                          85.4%; Score 35; DB 2; Length 15;
  Query Match
                          100.0%; Pred. No. 2.2;
  Best Local Similarity
            7; Conservative 0; Mismatches
                                                                             0;
                                                   0; Indels
                                                                 0; Gaps
  Matches
            1 LVFFAED 7
Qу
              111111
            2 LVFFAED 8
RESULT 36
ABG71014
     ABG71014 standard; peptide; 15 AA.
ID
XX
AC
     ABG71014;
XX
DΤ
     05-DEC-2002 (first entry)
XX
     Long form beta-amyloid protein fragment #10.
DΕ
```

XX Beta-amyloid; amyloid modulator; amyloidogenic protein; amyloidosis; KW familial amyloid polyneuropathy; familial amyloid cardiomyopathy; KW isolated cardiac amyloid; systemic senile amyloidosis; scrapie; myeloma; KW bovine spongiform encephalopathy; BSE; Creutzfeldt-Jakob disease; KW adult onset diabetes; Gerstmann-Straussler-Scheinker syndrome; KW insulinoma; atrial amyloidosis; idiopathic amyloidosis; haemodialysis; ΚW macroglobulinaemia-associated amyloidosis; reactive amyloidosis; ΚW primary localised cutaneous nodular amyloidosis; Sjogren's syndrome; KW hereditary cerebral haemorrhage with amyloidosis; Muckle-Wells syndrome; KW hereditary non-neuropathic systemic amyloidosis; KW familial Mediterranean Fever. ΚW XX Homo sapiens. OS XX PNUS2002098173-A1. XX 25-JUL-2002. PDXX 04-OCT-2001; 2001US-00972475. PFXX 95US-00404831. PR 14-MAR-1995; 95US-00475579. PR07-JUN-1995; 95US-00548998. PR 27-OCT-1995; 14-MAR-1996; 96US-00617267. PR XX (PRAE-) PRAECIS PHARM INC. PΑ XX Findeis MA, Benjamin H, Garnick MB, Gefter ML, Hundal A; PIKasman L, Musso G, Signer ER, Wakefield J, Reed MJ; PΙ XX DR WPI; 2002-697709/75. XX Amyloid modulator useful for treating a disorder associated with PTamyloidosis, comprises an amyloidogenic protein and/or a peptide fragment PTPTcoupled to a modifying group. XX Example 12; Page 35; 41pp; English. PS XX The invention describes an amyloid modulator comprising an amyloidogenic CC protein and/or peptide fragment coupled to a modifying group so that the CC compound modulates the aggregation of natural amyloid proteins or CC peptides. The modulator is used for treating a disorder associated with CC amyloidosis e.g. familial amyloid polyneuropathy (Portuguese, Japanese CC and Swedish types), familial amyloid cardiomyopathy (Danish type), CC isolated cardiac amyloid, systemic senile amyloidosis, scrapie, bovine CC spongiform encephalopathy, Creutzfeldt-Jakob disease, adult onset CC diabetes, Gerstmann-Straussler-Scheinker syndrome, insulinoma, isolated CCatrial amyloidosis, idiopathic (primary) amyloidosis, myeloma or CC macroglobulinaemia-associated amyloidosis, primary localised cutaneous CC nodular amyloidosis associated with Sjogren's syndrome, reactive CC (secondary) amyloidosis, familial Mediterranean Fever and familial CCamyloid nephropathy with urticaria and deafness (Muckle-Wells syndrome), CC hereditary cerebral haemorrhage with amyloidosis of Icelandic type, CC amyloidosis associated with long term haemodialysis, hereditary non-

neuropathic systemic amyloidosis (familial amyloid polyneuropathy III),

familial amyloidosis of Finnish type, amyloidosis associated with

CC

CC

```
medullary carcinoma of the thyroid, fibrinogen-associated hereditary
CC
     renal amyloidosis and lysozyme-associated hereditary systemic
CC
     amyloidosis. The compound is capable of altering and inhibiting beta-
CC
CC
     amyloid protein (beta-AP) aggregation of natural amyloidogenic proteins
     or peptides when contacted with a molar excess amount of natural beta-APs
CC
CC
     relative to the modulator. This sequence represents a fragment of the
CC
     long form of beta-amyloid used in the creation of an amyloid modulator
XX
SQ
     Sequence 15 AA;
                          85.4%; Score 35; DB 5; Length 15;
  Query Match
                          100.0%; Pred. No. 2.2;
  Best Local Similarity
            7; Conservative 0; Mismatches
                                                                             0;
                                                   0; Indels
                                                                 0; Gaps
            1 LVFFAED 7
Qy
              111111
            2 LVFFAED 8
RESULT 37
ABB05162
     ABB05162 standard; peptide; 15 AA.
XX
     ABB05162;
AC
XX
DT
     02-APR-2002 (first entry)
XX
DΕ
     Beta amyloid peptide (14-30) SEQ ID NO:14.
XX
     Beta amyloid peptide; beta-AP; beta amyloid precursor protein; A-beta;
KW
     APP-770; amyloid aggregation; amyloidogenic; Alzheimer's disease;
KW
     nootropic; neuroprotective; immunosuppressive; antimicrobial; auditory;
KW
     antidiabetic; antipyretic; dermatological; cardiovascular; nephrotropic;
KW
     amyloid aggregation inhibitor; neurotoxicity inhibitor; Down's syndrome;
KW
     amyloidogenic disease; beta amyloid deposition; amyloidosis;
KW
     hereditary cerebral haemorrhage; familial amyloid polyneuropathy.
KW
XX
OS
     Homo sapiens.
     Synthetic.
OS
XX
     US6319498-B1.
PN
XX
     20-NOV-2001.
PD
XX
                    96US-00617267.
PF
     14-MAR-1996;
XX
     14-MAR-1995;
                    95US-00404831.
PR
                    95US-00475579.
     07-JUN-1995;
PR
                    95US-00548998.
     27-OCT-1995;
PR
XX
     (PRAE-) PRAECIS PHARM INC.
PΑ
XX
     Findeis MA, Benjamin H, Garnick MB, Gefter ML, Hundal A;
PΙ
PΙ
     Kasman L, Musso G, Signer ER, Wakefield J, Reed MJ;
XX
     WPI; 2002-146668/19.
DR
XX
```

```
Amyloid modulator compound useful for treatment of an amyloidogenic
PΤ
     disease such as Alzheimer's disease comprises an aggregation core domain
PT
     and a modifying group attached to it.
PΤ
XX
PS
     Disclosure; Col 67; 54pp; English.
XX
     The present invention describes an amyloid modulator compound (I)
CC
     comprising an aggregation core domain and a modifying group attached to
CC
     it. (I) has nootropic, neuroprotective, immunosuppressive, antimicrobial,
CC
     antidiabetic, antipyretic, dermatological, cardiovascular, nephrotropic
CC
     and auditory activities, and can be used as a natural amyloid aggregation
CC
     inhibitor and a neurotoxicity inhibitor of natural beta amyloid peptide
CC
     (beta-AP). (I) are used in the manufacture of a medicament for the
CC
     diagnosis or treatment of an amyloidogenic disease e.g. Alzheimer's
CC
     disease and other clinical occurrences of beta amyloid deposition such as
CC
     Down's syndrome individuals and in patients with hereditary cerebral
CC
     haemorrhage with amyloidosis, and for treating a disorder associated with
CC
     amyloidosis such as familial amyloid polyneuropathy. (I) reduces the
CC
     toxicity of natural beta-AP aggregates to cultured neuronal cells. (I)
CC
     not only reduces the formation of neurotoxic aggregates but also have the
CC
     ability to reduce the neurotoxicity of performed A-beta fibrils. The
CC
     present sequence represents a beta-AP peptide, which is used in the
CC
     exemplification of the present invention
CC
XX
SQ
     Sequence 15 AA;
                          85.4%; Score 35; DB 5; Length 15;
  Query Match
                          100.0%; Pred. No. 2.2;
  Best Local Similarity
                                                                              0;
             7; Conservative
                                0; Mismatches
                                                    0; Indels
                                                                  0; Gaps
            1 LVFFAED 7
Qу
              111111
Db
            2 LVFFAED 8
RESULT 38
AAE26271
ID
     AAE26271 standard; peptide; 15 AA.
XX
     AAE26271;
AC
XX
DT
     14-NOV-2002
                  (first entry)
XX
     Human beta-amyloid peptide (beta-AP) #4.
DE
XX
     Human; amyloidogenic protein; Alzheimer's disease; Huntington's disease;
KW
     spongiform encephalopathy; familial amyloid cardiomyopathy; amyloidosis;
KW
     Gerstmann-Straussler-Scheinker syndrome; spongiform encephalopathy; GSS;
KW
     Creutzfeldt-Jacob disease; insulinoma; diabetes; body myocytis; myeloma;
KW
KW
     CJ; beta-amyloid; beta-AP.
XX
OS
     Homo sapiens.
XX
ΡN
     WO200242462-A2.
XX
PD
     30-MAY-2002.
XX
```

```
PF
     27-NOV-2001; 2001WO-US044581.
XX
PR
     27-NOV-2000; 2000US-0253302P.
     29-NOV-2000; 2000US-0250198P.
PR
     20-DEC-2000; 2000US-0257186P.
PR
XX
PΑ
     (PRAE-) PRAECIS PHARM INC.
XX
     Gefter ML, Israel DI, Joyal JL, Gosselin M;
PI
XX
     WPI; 2002-636427/68.
DR
XX
     Novel therapeutic agent useful for treating an amyloidogenic disorder,
PT
     e.g. Alzheimer's disease, comprises an immunoglobulin heavy chain
PT
     constant region linked to a peptide capable of binding amyloidogenic
PT
PT
     protein.
XX
     Example 8; Page 76; 79pp; English.
PS
XX
     The invention relates to a compound comprising an immunoglobulin (Ig)
CC
     heavy chain constant region or its fragment that retains the ability to
CC
     bind an Fc receptor linked by a linker group or a direct bond to a
CC
     peptide capable of binding an amyloidogenic protein. The invention is
CC
     useful for clearing an amyloidogenic protein such as beta-amyloid,
CC
CC
     transthyretin (TTR), prion protein (PrP), islet amyloid polypeptide
     (IAPP), atrial natriuretic factor (ANF), kappa light chain, lambda light
CC
CC
     chain, amyloid A, procalcitonin, cystatin C, beta2-microglobulin, ApoA-I,
CC
     gelsolin, calcitonin, fibrinogen, Huntington, alpha-synuclein and
CC
     lysozyme from a subject and for treating an amyloidogenic disorder such
     as Alzheimer's disease and spongiform encephalopathy. Disorders treatable
CC
     include those caused or characterised by deposits of TTR (eg. familial
CC
     amyloid cardiomyopathy), PrP (eg. spongiform encephalopathies, including
CC
     scrapie in sheep, bovine spongiform encephalopathy in cows and
CC
     Creutzfeldt-Jacob disease (CJ) and Gerstmann-Straussler-Scheinker
CC
     syndrome (GSS) in humans), IAPP (eg. insulinoma, adult onset diabetes),
CC
     ANF (eg. isolated atrial amyloid), kappa or lambda light chain (eg.
CC
     idiopathic amyloidosis, myeloma), amyloid A (eg. amyloidosis), Apo A-I
CC
     (eq. hereditary non-neuropathic systemic amyloidosis), Gelsolin (eq.
CC
     familial amyloidosis of Finnish type), Fibrinogen (eg. hereditary renal
CC
     amyloidosis), Lysozyme (eq. hereditary systemic amyloidosis). Other
CC
     examples of amyloidogenic disorders include Huntington's disease and
CC
     inclusion body myocytis. The present sequence is human beta-amyloid
CC
CC
     peptide (beta-AP)
XX
SO
     Sequence 15 AA;
                          85.4%; Score 35; DB 5; Length 15;
  Query Match
                          100.0%; Pred. No. 2.2;
  Best Local Similarity
                                                                              0;
             7; Conservative
                                                   0; Indels
                                                                  0; Gaps
  Matches
                               0; Mismatches
Qу
            1 LVFFAED 7
              +1111111
            2 LVFFAED 8
Db
```

ID ABU79057 standard; peptide; 15 AA. XX AC ABU79057; XX 17-JUN-2003 (first entry) DTXX Aggregation blocking peptide #9. DE XX Amyloid formation; amyloid-like deposit; Alzheimer's disease; KW pathological beta-sheet-rich conformation; Down's syndrome; KW amyloidosis disorder; human prion disease; kuru; CJD; ΚW Creutzfeldt-Jakob disease; Gerstmann-Straussler-Scheinker syndrome; GSS; KW prion associated human neurodegenerative disease; animal prion disease; KW scrapie; spongiform encephalopathy; transmissible mink encephalopathy; KW chronic wasting disease. KW XX Unidentified. OS XX PN US6462171-B1. XX PD 08-OCT-2002. XX 96US-00766596. PF12-DEC-1996; XX 07-JUN-1995; 95US-00478326. PR 10-APR-1996; 96US-00630645. PR XX (UYNY) UNIV NEW YORK STATE. PAXX Soto-Jara C, Baumann MH, Frangione B; PΙ XX WPI; 2003-379012/36. DR XX Novel inhibitory peptides which inhibit and structurally block abnormal PTfolding of protein into amyloid or amyloid-like deposit and into PTpathological beta-sheet rich conformation, useful for treating PTPTAlzheimer's disease. XX Disclosure; Col 49-50; 51pp; English. PS XX The invention describes an isolated inhibitory peptide (I) which CC interacts with a hydrophobic beta-sheet forming cluster of amino acid CC residues on a protein or peptide for amyloid or amyloid-like deposit CC formation, and inhibits or structurally blocks the abnormal folding of CCproteins and peptides into amyloid or amyloid-like deposits and into CC pathological beta-sheet-rich conformation. (I) is useful for disorders or CC diseases associated with abnormal protein folding into amyloid or amyloid CC -like deposits or into pathological beta-sheet-rich precursors of such CCdeposits, such as Alzheimer's disease, Down's syndrome, other amyloidosis CC disorders, human prion diseases, such as kuru, Creutzfeldt-Jakob disease CC (CJD), Gerstmann-Straussler-Scheinker syndrome (GSS), prion associated CC CC human neurodegenerative diseases as well as animal prion diseases such as scrapie, spongiform encephalopathy, transmissible mink encephalopathy and CC chronic wasting disease of mule deer and elk. (I) is also useful for CC detecting and diagnosing the presence or absence of amyloid or amyloid-CC like deposits in vivo and its precursors. This is the amino acid sequence CC of peptide associated with the inhibition of amyloid or amyloid like CC

```
CC
     deposits
XX
SQ
     Sequence 15 AA;
                          85.4%; Score 35; DB 6; Length 15;
  Query Match
                          100.0%; Pred. No. 2.2;
  Best Local Similarity
          7; Conservative
                                 0; Mismatches
                                                    0; Indels
                                                                  0; Gaps
                                                                              0;
 Matches
            1 LVFFAED 7
Qу
              1111111
            6 LVFFAED 12
Db
RESULT 40
ABU79064
     ABU79064 standard; peptide; 15 AA.
XX
AC
     ABU79064;
XX
     17-JUN-2003 (first entry)
DT
XX
     Aggregation blocking peptide #16.
DE
XX
     Amyloid formation; amyloid-like deposit; Alzheimer's disease;
KW
     pathological beta-sheet-rich conformation; Down's syndrome;
KW
     amyloidosis disorder; human prion disease; kuru; CJD;
KW
     Creutzfeldt-Jakob disease; Gerstmann-Straussler-Scheinker syndrome; GSS;
KW
     prion associated human neurodegenerative disease; animal prion disease;
KW
     scrapie; spongiform encephalopathy; transmissible mink encephalopathy;
KW
KW
     chronic wasting disease.
XX
OS
     Unidentified.
XX
PN
     US6462171-B1.
XX
     08-OCT-2002.
PD
XX
                    96US-00766596.
PF
     12-DEC-1996;
XX
                    95US-00478326.
PR
     07-JUN-1995;
                    96US-00630645.
     10-APR-1996;
PR
XX
     (UYNY ) UNIV NEW YORK STATE.
PA
XX
     Soto-Jara C, Baumann MH, Frangione B;
PΙ
XX
     WPI; 2003-379012/36.
DR
XX
     Novel inhibitory peptides which inhibit and structurally block abnormal
PT
     folding of protein into amyloid or amyloid-like deposit and into
PT
     pathological beta-sheet rich conformation, useful for treating
PT
PT
     Alzheimer's disease.
XX
     Disclosure; Col 51-52; 51pp; English.
PS
XX
     The invention describes an isolated inhibitory peptide (I) which
CC
     interacts with a hydrophobic beta-sheet forming cluster of amino acid
CC
```

```
residues on a protein or peptide for amyloid or amyloid-like deposit
CC
     formation, and inhibits or structurally blocks the abnormal folding of
CC
     proteins and peptides into amyloid or amyloid-like deposits and into
CC
     pathological beta-sheet-rich conformation. (I) is useful for disorders or
CC
     diseases associated with abnormal protein folding into amyloid or amyloid
CC
CC
     -like deposits or into pathological beta-sheet-rich precursors of such
     deposits, such as Alzheimer's disease, Down's syndrome, other amyloidosis
CC
     disorders, human prion diseases, such as kuru, Creutzfeldt-Jakob disease
CC
     (CJD), Gerstmann-Straussler-Scheinker syndrome (GSS), prion associated
CC
     human neurodegenerative diseases as well as animal prion diseases such as
CC
CC
     scrapie, spongiform encephalopathy, transmissible mink encephalopathy and
     chronic wasting disease of mule deer and elk. (I) is also useful for
CC
     detecting and diagnosing the presence or absence of amyloid or amyloid-
CC
     like deposits in vivo and its precursors. This is the amino acid sequence
CC
     of peptide associated with the inhibition of amyloid or amyloid like
CC
CC
     deposits
XX
     Sequence 15 AA;
SO
                          85.4%; Score 35; DB 6;
                                                    Length 15;
  Query Match
                          100.0%; Pred. No. 2.2;
  Best Local Similarity
                                                                              0;
                                0; Mismatches
             7; Conservative
                                                   0; Indels
                                                                 0; Gaps
            1 LVFFAED 7
Qу
              6 LVFFAED 12
RESULT 41
ABU79058
     ABU79058 standard; peptide; 15 AA.
ID
XX
AC
     ABU79058;
XX
DT
     17-JUN-2003 (first entry)
XX
     Aggregation blocking peptide #10.
DE
XX
     Amyloid formation; amyloid-like deposit; Alzheimer's disease;
KW
     pathological beta-sheet-rich conformation; Down's syndrome;
KW
     amyloidosis disorder; human prion disease; kuru; CJD;
KW
     Creutzfeldt-Jakob disease; Gerstmann-Straussler-Scheinker syndrome; GSS;
KW
     prion associated human neurodegenerative disease; animal prion disease;
KW
     scrapie; spongiform encephalopathy; transmissible mink encephalopathy;
KW
     chronic wasting disease.
KW
XX
OS
     Unidentified.
XX
PN
     US6462171-B1.
XX
     08-OCT-2002.
PD
XX
                    96US-00766596.
PF
     12-DEC-1996;
XX
     07-JUN-1995;
                    95US-00478326.
PR
     10-APR-1996;
                    96US-00630645.
PR
XX
```

```
PA
     (UYNY ) UNIV NEW YORK STATE.
XX
PΙ
     Soto-Jara C, Baumann MH,
                                Frangione B;
XX
     WPI; 2003-379012/36.
DR
XX
     Novel inhibitory peptides which inhibit and structurally block abnormal
PT
     folding of protein into amyloid or amyloid-like deposit and into
PT
     pathological beta-sheet rich conformation, useful for treating
PT
     Alzheimer's disease.
PT
XX
     Disclosure; Col 49-50; 51pp; English.
PS
XX
     The invention describes an isolated inhibitory peptide (I) which
CC
     interacts with a hydrophobic beta-sheet forming cluster of amino acid
CC
     residues on a protein or peptide for amyloid or amyloid-like deposit
CC
     formation, and inhibits or structurally blocks the abnormal folding of
CC
     proteins and peptides into amyloid or amyloid-like deposits and into
CC
     pathological beta-sheet-rich conformation. (I) is useful for disorders or
CC
     diseases associated with abnormal protein folding into amyloid or amyloid
CC
     -like deposits or into pathological beta-sheet-rich precursors of such
CC
     deposits, such as Alzheimer's disease, Down's syndrome, other amyloidosis
CC
     disorders, human prion diseases, such as kuru, Creutzfeldt-Jakob disease
CC
     (CJD), Gerstmann-Straussler-Scheinker syndrome (GSS), prion associated
CC
     human neurodegenerative diseases as well as animal prion diseases such as
CC
     scrapie, spongiform encephalopathy, transmissible mink encephalopathy and
CC
     chronic wasting disease of mule deer and elk. (I) is also useful for
CC
     detecting and diagnosing the presence or absence of amyloid or amyloid-
CC
     like deposits in vivo and its precursors. This is the amino acid sequence
CC
     of peptide associated with the inhibition of amyloid or amyloid like
CC
     deposits
CC
XX
SQ
     Sequence 15 AA;
                          85.4%; Score 35; DB 6; Length 15;
  Query Match
                          100.0%; Pred. No. 2.2;
  Best Local Similarity
                                                                      Gaps
                                                                              0;
                                                   0; Indels
                                                                 0;
             7; Conservative
                                 0; Mismatches
  Matches
            1 LVFFAED 7
Qу
              ++++++
            6 LVFFAED 12
Db
RESULT 42
ABU79055
     ABU79055 standard; peptide; 15 AA.
XX
AC
     ABU79055;
XX
DT
     17-JUN-2003 (first entry)
XX
DE
     Aggregation blocking peptide #7.
XX
     Amyloid formation; amyloid-like deposit; Alzheimer's disease;
KW
     pathological beta-sheet-rich conformation; Down's syndrome;
KW
     amyloidosis disorder; human prion disease; kuru; CJD;
KW
     Creutzfeldt-Jakob disease; Gerstmann-Straussler-Scheinker syndrome; GSS;
KW
```

```
prion associated human neurodegenerative disease; animal prion disease;
KW
     scrapie; spongiform encephalopathy; transmissible mink encephalopathy;
KW
KW
     chronic wasting disease.
XX
OS
     Unidentified.
XX
PN
     US6462171-B1.
XX
PD
     08-OCT-2002.
XX
     12-DEC-1996;
                    96US-00766596.
PF
XX
                    95US-00478326.
PR
     07-JUN-1995;
                    96US-00630645.
PR
     10-APR-1996;
XX
     (UYNY ) UNIV NEW YORK STATE.
PA
XX
                  Baumann MH,
                                Frangione B;
PI
     Soto-Jara C,
XX
     WPI; 2003-379012/36.
DR
XX
     Novel inhibitory peptides which inhibit and structurally block abnormal
PТ
PT
     folding of protein into amyloid or amyloid-like deposit and into
PT
     pathological beta-sheet rich conformation, useful for treating
РT
     Alzheimer's disease.
XX
PS
     Disclosure; Col 49-50; 51pp; English.
XX
     The invention describes an isolated inhibitory peptide (I) which
CC
     interacts with a hydrophobic beta-sheet forming cluster of amino acid
CC
     residues on a protein or peptide for amyloid or amyloid-like deposit
CC
     formation, and inhibits or structurally blocks the abnormal folding of
CC
     proteins and peptides into amyloid or amyloid-like deposits and into
CC
     pathological beta-sheet-rich conformation. (I) is useful for disorders or
CC
     diseases associated with abnormal protein folding into amyloid or amyloid
CC
     -like deposits or into pathological beta-sheet-rich precursors of such
CC
     deposits, such as Alzheimer's disease, Down's syndrome, other amyloidosis
CC
     disorders, human prion diseases, such as kuru, Creutzfeldt-Jakob disease
CC
     (CJD), Gerstmann-Straussler-Scheinker syndrome (GSS), prion associated
CC
     human neurodegenerative diseases as well as animal prion diseases such as
CC
     scrapie, spongiform encephalopathy, transmissible mink encephalopathy and
CC
     chronic wasting disease of mule deer and elk. (I) is also useful for
CC
     detecting and diagnosing the presence or absence of amyloid or amyloid-
CC
     like deposits in vivo and its precursors. This is the amino acid sequence
CC
     of peptide associated with the inhibition of amyloid or amyloid like
CC
CC
     deposits
XX
SQ
     Sequence 15 AA;
                                  Score 35; DB 6;
                                                    Length 15;
  Query Match
                           85.4%;
                          100.0%; Pred. No. 2.2;
  Best Local Similarity
                                                                      Gaps
                                                                              0;
  Matches
             7; Conservative
                                 0; Mismatches
                                                    0; Indels
            1 LVFFAED 7
Qу
              111111
            6 LVFFAED 12
Db
```

```
RESULT 43
ABU79056
    ABU79056 standard; peptide; 15 AA.
ID
XX
AC
     ABU79056;
XX
DT
    17-JUN-2003 (first entry)
XX
     Aggregation blocking peptide #8.
DE
XX
     Amyloid formation; amyloid-like deposit; Alzheimer's disease;
KW
     pathological beta-sheet-rich conformation; Down's syndrome;
KW
     amyloidosis disorder; human prion disease; kuru; CJD;
KW
KW
     Creutzfeldt-Jakob disease; Gerstmann-Straussler-Scheinker syndrome; GSS;
     prion associated human neurodegenerative disease; animal prion disease;
KW
     scrapie; spongiform encephalopathy; transmissible mink encephalopathy;
KW
KW
     chronic wasting disease.
XX
OS
     Unidentified.
XX
PN
     US6462171-B1.
XX
PD
     08-OCT-2002.
XX
PF
     12-DEC-1996;
                    96US-00766596.
XX
     07-JUN-1995;
PR
                    95US-00478326.
PR
     10-APR-1996;
                    96US-00630645.
XX
     (UYNY ) UNIV NEW YORK STATE.
PA
XX
     Soto-Jara C, Baumann MH, Frangione B;
PI
XX
DR
     WPI; 2003-379012/36.
XX
     Novel inhibitory peptides which inhibit and structurally block abnormal
PT
     folding of protein into amyloid or amyloid-like deposit and into
PT
     pathological beta-sheet rich conformation, useful for treating
PT
PT
     Alzheimer's disease.
XX
     Disclosure; Col 49-50; 51pp; English.
PS
XX
     The invention describes an isolated inhibitory peptide (I) which
CC
     interacts with a hydrophobic beta-sheet forming cluster of amino acid
CC
     residues on a protein or peptide for amyloid or amyloid-like deposit
CC
     formation, and inhibits or structurally blocks the abnormal folding of
CC
     proteins and peptides into amyloid or amyloid-like deposits and into
CC
     pathological beta-sheet-rich conformation. (I) is useful for disorders or
CC
     diseases associated with abnormal protein folding into amyloid or amyloid
CC
     -like deposits or into pathological beta-sheet-rich precursors of such
CC
     deposits, such as Alzheimer's disease, Down's syndrome, other amyloidosis
CC
     disorders, human prion diseases, such as kuru, Creutzfeldt-Jakob disease
CC
     (CJD), Gerstmann-Straussler-Scheinker syndrome (GSS), prion associated
CC
CC
     human neurodegenerative diseases as well as animal prion diseases such as
     scrapie, spongiform encephalopathy, transmissible mink encephalopathy and
CC
     chronic wasting disease of mule deer and elk. (I) is also useful for
CC
```

```
detecting and diagnosing the presence or absence of amyloid or amyloid-
CC
    like deposits in vivo and its precursors. This is the amino acid sequence
CC
     of peptide associated with the inhibition of amyloid or amyloid like
CC
CC
    deposits
XX
     Sequence 15 AA;
SO
                          85.4%; Score 35; DB 6; Length 15;
 Query Match
                          100.0%; Pred. No. 2.2;
  Best Local Similarity
            7; Conservative
                               0; Mismatches
                                                   0; Indels
                                                                  0; Gaps
                                                                              0;
 Matches
            1 LVFFAED 7
Qу
              6 LVFFAED 12
Db
RESULT 44
ABU79062
    ABU79062 standard; peptide; 15 AA.
ID
XX
AC
    ABU79062;
XX
    17-JUN-2003 (first entry)
DT
XX
DE
    Aggregation blocking peptide #14.
XX
     Amyloid formation; amyloid-like deposit; Alzheimer's disease;
KW
     pathological beta-sheet-rich conformation; Down's syndrome;
KW
     amyloidosis disorder; human prion disease; kuru; CJD;
KW
     Creutzfeldt-Jakob disease; Gerstmann-Straussler-Scheinker syndrome; GSS;
KW
     prion associated human neurodegenerative disease; animal prion disease;
KW
     scrapie; spongiform encephalopathy; transmissible mink encephalopathy;
KW
     chronic wasting disease.
KW
XX
OS
     Unidentified.
XX
PN
     US6462171-B1.
XX
     08-OCT-2002.
PD
XX
                    96US-00766596.
PF
     12-DEC-1996;
XX
     07-JUN-1995;
                    95US-00478326.
PR
     10-APR-1996;
                    96US-00630645.
PR
XX
PA
     (UYNY ) UNIV NEW YORK STATE.
XX
     Soto-Jara C, Baumann MH, Frangione B;
PI
XX
DR
     WPI; 2003-379012/36.
XX
     Novel inhibitory peptides which inhibit and structurally block abnormal
PT
     folding of protein into amyloid or amyloid-like deposit and into
PT
PT
     pathological beta-sheet rich conformation, useful for treating
PT
     Alzheimer's disease.
XX
     Disclosure; Col 51-52; 51pp; English.
PS
```

```
XX
     The invention describes an isolated inhibitory peptide (I) which
CC
     interacts with a hydrophobic beta-sheet forming cluster of amino acid
CC
     residues on a protein or peptide for amyloid or amyloid-like deposit
CC
CC
     formation, and inhibits or structurally blocks the abnormal folding of
CC
     proteins and peptides into amyloid or amyloid-like deposits and into
CC
     pathological beta-sheet-rich conformation. (I) is useful for disorders or
     diseases associated with abnormal protein folding into amyloid or amyloid
CC
     -like deposits or into pathological beta-sheet-rich precursors of such
CC
CC
     deposits, such as Alzheimer's disease, Down's syndrome, other amyloidosis
CC
     disorders, human prion diseases, such as kuru, Creutzfeldt-Jakob disease
     (CJD), Gerstmann-Straussler-Scheinker syndrome (GSS), prion associated
CC
     human neurodegenerative diseases as well as animal prion diseases such as
CC
CC
     scrapie, spongiform encephalopathy, transmissible mink encephalopathy and
CC
     chronic wasting disease of mule deer and elk. (I) is also useful for
     detecting and diagnosing the presence or absence of amyloid or amyloid-
CC
     like deposits in vivo and its precursors. This is the amino acid sequence
CC
CC
     of peptide associated with the inhibition of amyloid or amyloid like
CC
     deposits
XX
     Sequence 15 AA;
SO
  Query Match
                          85.4%; Score 35; DB 6; Length 15;
                          100.0%; Pred. No. 2.2;
  Best Local Similarity
                                                                              0;
  Matches
            7; Conservative 0; Mismatches
                                                   0; Indels
                                                                  0; Gaps
Qу
            1 LVFFAED 7
              111111
            6 LVFFAED 12
Db
RESULT 45
ABW00192
     ABW00192 standard; peptide; 15 AA.
ΙD
XX
AC
     ABW00192;
XX
DT
     15-JAN-2004 (first entry)
XX
     Peptide #10 used in the invention.
DE
XX
     Amyloid-like fibril deposit; prion related encephalopathy; gene therapy;
KW
     Alzheimer's disease.
KW
XX
     Unidentified.
OS
XX
     US2003087407-A1.
PN
XX
PD
     08-MAY-2003.
XX
PF
     06-SEP-2002; 2002US-00235483.
XX
PR
     07-JUN-1995;
                    95US-00478326.
PR
     10-APR-1996;
                    96US-00630645.
                    96US-00766596.
PR
     12-DEC-1996;
XX
     (UYNY ) UNIV NEW YORK STATE.
PΑ
```

```
XX
PI
     Soto-Jara C, Baumann MH,
                               Frangione B;
XX
DR
     WPI; 2003-616149/58.
XX
PΤ
     New inhibitory peptide, useful for preparing a composition for
PT
     diagnosing, preventing or treating disorders associated with amyloid-like
     fibril deposits, e.g. Alzheimer's disease, or prion related
PT
     encephalopathies.
PT
XX
     Claim 1; Page 27; 52pp; English.
PS
XX
     The invention relates to inhibitory peptide comprising a portion of at
CC
     least three amino acid residues and a sequence predicted not to adopt a
CC
     beta-sheet structure that associates with a hydrophobic beta-sheet
CC
     cluster on a protein or peptide involved in the abnormal folding into a
CC
     beta-sheet structure, to structurally block the abnormal folding of the
CC
     protein or peptide. The inhibitory peptide is useful for preparing a
CC
     composition for preventing, treating or detecting disorders or diseases
CC
     associated with amyloid-like fibril deposits e.g. Alzheimer's disease and
CC
     prion related encephalopathies. The invention is also useful in gene
CC
     therapy. The present sequence is a peptide used in the invention
CC
XX
SQ
     Sequence 15 AA;
                          85.4%; Score 35; DB 7; Length 15;
  Query Match
                          100.0%; Pred. No. 2.2;
  Best Local Similarity
                                                                 0; Gaps
                                                                              0:
  Matches
            7; Conservative
                               0; Mismatches
                                                   0; Indels
Qу
            1 LVFFAED 7
              6 LVFFAED 12
Dh
RESULT 46
ABW00190
     ABW00190 standard; peptide; 15 AA.
ID
XX
AC
     ABW00190;
XX
DT
     15-JAN-2004 (first entry)
XX
DΕ
     Peptide #8 used in the invention.
XX
     Amyloid-like fibril deposit; prion related encephalopathy; gene therapy;
KW
     Alzheimer's disease.
KW
XX
OS
     Unidentified.
XX
     US2003087407-A1.
PN
XX
PD
     08-MAY-2003.
XX
PF
     06-SEP-2002; 2002US-00235483.
XX
                    95US-00478326.
PR
     07-JUN-1995;
                    96US-00630645.
     10-APR-1996;
PR
```

```
96US-00766596.
PR
     12-DEC-1996;
XX
PA
     (UYNY ) UNIV NEW YORK STATE.
XX
     Soto-Jara C, Baumann MH, Frangione B;
PI
XX
DR
     WPI; 2003-616149/58.
XX
     New inhibitory peptide, useful for preparing a composition for
РΤ
     diagnosing, preventing or treating disorders associated with amyloid-like
PT
     fibril deposits, e.g. Alzheimer's disease, or prion related
РT
PT
     encephalopathies.
XX
     Claim 1; Page 26; 52pp; English.
PS
XX
     The invention relates to inhibitory peptide comprising a portion of at
CC
     least three amino acid residues and a sequence predicted not to adopt a
CC
     beta-sheet structure that associates with a hydrophobic beta-sheet
CC
     cluster on a protein or peptide involved in the abnormal folding into a
CC
     beta-sheet structure, to structurally block the abnormal folding of the
CC
     protein or peptide. The inhibitory peptide is useful for preparing a
CC
     composition for preventing, treating or detecting disorders or diseases
CC
CC
     associated with amyloid-like fibril deposits e.g. Alzheimer's disease and
CC
     prion related encephalopathies. The invention is also useful in gene
     therapy. The present sequence is a peptide used in the invention
CC
XX
SO
     Sequence 15 AA;
                          85.4%; Score 35; DB 7; Length 15;
  Query Match
                          100.0%; Pred. No. 2.2;
  Best Local Similarity
             7; Conservative
                                0; Mismatches
                                                   0; Indels
                                                                 0; Gaps
                                                                              0;
  Matches
            1 LVFFAED 7
Qу
              6 LVFFAED 12
RESULT 47
ABW00198
     ABW00198 standard; peptide; 15 AA.
ΙD
XX
AC
     ABW00198;
XX
DT
     15-JAN-2004 (first entry)
XX
     Peptide #16 used in the invention.
DE
XX
     Amyloid-like fibril deposit; prion related encephalopathy; gene therapy;
KW
     Alzheimer's disease.
KW
XX
     Unidentified.
OS
XX
PN
     US2003087407-A1.
XX
PD
     08-MAY-2003.
XX
     06-SEP-2002; 2002US-00235483.
PF
```

```
XX
PR
     07-JUN-1995;
                     95US-00478326.
PR
     10-APR-1996;
                    96US-00630645.
     12-DEC-1996;
                    96US-00766596.
PR
XX
      (UYNY ) UNIV NEW YORK STATE.
 PΑ
XX
     Soto-Jara C, Baumann MH, Frangione B;
 PΙ
XX
     WPI; 2003-616149/58.
 DR
XX
     New inhibitory peptide, useful for preparing a composition for
 PT
     diagnosing, preventing or treating disorders associated with amyloid-like
 PT
     fibril deposits, e.g. Alzheimer's disease, or prion related
 PТ
 PT
     encephalopathies.
XX
 PS
     Claim 1; Page 28; 52pp; English.
XX
     The invention relates to inhibitory peptide comprising a portion of at
 CC
     least three amino acid residues and a sequence predicted not to adopt a
 CC
     beta-sheet structure that associates with a hydrophobic beta-sheet
 CC
     cluster on a protein or peptide involved in the abnormal folding into a
 CC
     beta-sheet structure, to structurally block the abnormal folding of the
 CC
 CC
     protein or peptide. The inhibitory peptide is useful for preparing a
 CC
     composition for preventing, treating or detecting disorders or diseases
     associated with amyloid-like fibril deposits e.g. Alzheimer's disease and
 CC
 CC
     prion related encephalopathies. The invention is also useful in gene
 CC
     therapy. The present sequence is a peptide used in the invention
 XX
 SO
      Sequence 15 AA;
                           85.4%; Score 35; DB 7; Length 15;
  Query Match
                           100.0%; Pred. No. 2.2;
  Best Local Similarity
                                                                  0; Gaps
                                                                              0;
             7; Conservative 0; Mismatches
                                                    0; Indels
             1 LVFFAED 7
 Qу
               6 LVFFAED 12
Db
 RESULT 48
 ABW00189
      ABW00189 standard; peptide; 15 AA.
 XX
 AC
     ABW00189;
 XX
      15-JAN-2004 (first entry)
 DT
 XX
 DE
      Peptide #7 used in the invention.
 XX
      Amyloid-like fibril deposit; prion related encephalopathy; gene therapy;
 KW
 KW
      Alzheimer's disease.
 XX
 OS
      Unidentified.
 XX
      US2003087407-A1.
 PN
 XX
```

```
PD
     08-MAY-2003.
XX
PF
     06-SEP-2002; 2002US-00235483.
XX
PR
     07-JUN-1995;
                    95US-00478326.
PR
     10-APR-1996;
                    96US-00630645.
PR
     12-DEC-1996;
                    96US-00766596.
XX
     (UYNY ) UNIV NEW YORK STATE.
PΑ
XX
PI
     Soto-Jara C, Baumann MH, Frangione B;
XX
DR
     WPI; 2003-616149/58.
XX
PT
     New inhibitory peptide, useful for preparing a composition for
PT
     diagnosing, preventing or treating disorders associated with amyloid-like
     fibril deposits, e.g. Alzheimer's disease, or prion related
PT
PT
     encephalopathies.
XX
PS
     Claim 1; Page 26; 52pp; English.
XX
CC
     The invention relates to inhibitory peptide comprising a portion of at
CC
     least three amino acid residues and a sequence predicted not to adopt a
CC
     beta-sheet structure that associates with a hydrophobic beta-sheet
CC
     cluster on a protein or peptide involved in the abnormal folding into a
CC
     beta-sheet structure, to structurally block the abnormal folding of the
CC
     protein or peptide. The inhibitory peptide is useful for preparing a
    composition for preventing, treating or detecting disorders or diseases
CC
     associated with amyloid-like fibril deposits e.g. Alzheimer's disease and
CC
     prion related encephalopathies. The invention is also useful in gene
CC
CC
     therapy. The present sequence is a peptide used in the invention
XX
SQ
     Sequence 15 AA;
  Query Match
                          85.4%; Score 35; DB 7;
                                                    Length 15;
  Best Local Similarity
                          100.0%; Pred. No. 2.2;
            7; Conservative
                               0; Mismatches
                                                                 0; Gaps
                                                                              0:
  Matches
                                                   0; Indels
            1 LVFFAED 7
Qу
              1111111
            6 LVFFAED 12
Dh
RESULT 49
ABW00191
     ABW00191 standard; peptide; 15 AA.
ID
XX
     ABW00191;
AC
XX
     15-JAN-2004 (first entry)
DT
XX
     Peptide #9 used in the invention.
DE
XX
ΚW
     Amyloid-like fibril deposit; prion related encephalopathy; gene therapy;
ΚW
     Alzheimer's disease.
XX
OS
     Unidentified.
```

```
XX
PN
    US2003087407-A1.
XX
PD
     08-MAY-2003.
XX
PF
     06-SEP-2002; 2002US-00235483.
XX
PR
     07-JUN-1995;
                    95US-00478326.
PR
     10-APR-1996;
                    96US-00630645.
PR
     12-DEC-1996;
                    96US-00766596.
XX
PΑ
     (UYNY ) UNIV NEW YORK STATE.
XX
PΙ
     Soto-Jara C, Baumann MH, Frangione B;
XX
DR
    WPI; 2003-616149/58.
XX
PT
    New inhibitory peptide, useful for preparing a composition for
PT
    diagnosing, preventing or treating disorders associated with amyloid-like
PT
     fibril deposits, e.g. Alzheimer's disease, or prion related
PT
     encephalopathies.
XX
PS
    Claim 1; Page 26; 52pp; English.
XX
CC
    The invention relates to inhibitory peptide comprising a portion of at
    least three amino acid residues and a sequence predicted not to adopt a
CC
CC
    beta-sheet structure that associates with a hydrophobic beta-sheet
    cluster on a protein or peptide involved in the abnormal folding into a
CC
CC
    beta-sheet structure, to structurally block the abnormal folding of the
CC
    protein or peptide. The inhibitory peptide is useful for preparing a
     composition for preventing, treating or detecting disorders or diseases
CC
CC
     associated with amyloid-like fibril deposits e.g. Alzheimer's disease and
CC
     prion related encephalopathies. The invention is also useful in gene
CC
     therapy. The present sequence is a peptide used in the invention
XX
     Sequence 15 AA;
SO
                          85.4%; Score 35; DB 7; Length 15;
  Query Match
                          100.0%; Pred. No. 2.2;
  Best Local Similarity
 Matches
            7; Conservative 0; Mismatches
                                                   0; Indels
                                                                 0; Gaps
                                                                             0;
            1 LVFFAED 7
Qу
              111111
Db
            6 LVFFAED 12
RESULT 50
ABW00196
ID
    ABW00196 standard; peptide; 15 AA.
XX
AC
    ABW00196;
XX
DT
     15-JAN-2004 (first entry)
XX
DE
     Peptide #14 used in the invention.
XX
KW
     Amyloid-like fibril deposit; prion related encephalopathy; gene therapy;
```

```
KW
     Alzheimer's disease.
XX
OS
     Unidentified.
XX
PN
     US2003087407-A1.
XX
PD
     08-MAY-2003.
XX
     06-SEP-2002; 2002US-00235483.
PF
XX
PR
     07-JUN-1995;
                    95US-00478326.
PR
     10-APR-1996;
                    96US-00630645.
     12-DEC-1996;
                    96US-00766596.
PR
XX
PA
     (UYNY ) UNIV NEW YORK STATE.
XX
PΙ
     Soto-Jara C, Baumann MH, Frangione B;
XX
DR
     WPI: 2003-616149/58.
XX
PT
     New inhibitory peptide, useful for preparing a composition for
     diagnosing, preventing or treating disorders associated with amyloid-like
РT
PT
     fibril deposits, e.g. Alzheimer's disease, or prion related
PT
     encephalopathies.
XX
     Claim 1; Page 27; 52pp; English.
PS
XX
     The invention relates to inhibitory peptide comprising a portion of at
CC
CC
     least three amino acid residues and a sequence predicted not to adopt a
CC
     beta-sheet structure that associates with a hydrophobic beta-sheet
CC
     cluster on a protein or peptide involved in the abnormal folding into a
CC
     beta-sheet structure, to structurally block the abnormal folding of the
CC
     protein or peptide. The inhibitory peptide is useful for preparing a
CC
     composition for preventing, treating or detecting disorders or diseases
     associated with amyloid-like fibril deposits e.g. Alzheimer's disease and
CC
CC
     prion related encephalopathies. The invention is also useful in gene
CC
     therapy. The present sequence is a peptide used in the invention
XX
SQ
     Sequence 15 AA;
  Query Match
                          85.4%; Score 35; DB 7; Length 15;
                          100.0%; Pred. No. 2.2;
  Best Local Similarity
                                 0; Mismatches
                                                                              0;
 Matches
            7; Conservative
                                                   0; Indels
                                                                 0; Gaps
            1 LVFFAED 7
Qу
              6 LVFFAED 12
Db
RESULT 51
AAE26330
     AAE26330 standard; peptide; 16 AA.
XX
AC
     AAE26330;
XX
     14-NOV-2002 (first entry)
DT
XX
```

Human beta-amyloid peptide mutant (Abeta residues 10-25). DE XX Human; amyloidogenic protein; Alzheimer's disease; Huntington's disease; KW spongiform encephalopathy; familial amyloid cardiomyopathy; amyloidosis; KW Gerstmann-Straussler-Scheinker syndrome; spongiform encephalopathy; GSS; KW Creutzfeldt-Jacob disease; insulinoma; diabetes; body myocytis; myeloma; KW KW CJ; beta-amyloid; mutant; mutein. XX os Homo sapiens. OS Synthetic. XX W0200242462-A2. PNXX PD30-MAY-2002. XX 27-NOV-2001; 2001WO-US044581. PFXX PR 27-NOV-2000; 2000US-0253302P. PR 29-NOV-2000; 2000US-0250198P. 20-DEC-2000; 2000US-0257186P. PR XX (PRAE-) PRAECIS PHARM INC. PΑ XX Gefter ML, Israel DI, Joyal JL, Gosselin M; PIXX DR WPI; 2002-636427/68. XX Novel therapeutic agent useful for treating an amyloidogenic disorder, PTe.q. Alzheimer's disease, comprises an immunoglobulin heavy chain PTconstant region linked to a peptide capable of binding amyloidogenic PTPTprotein. XX PS Claim 18; Page; 79pp; English. XX CC The invention relates to a compound comprising an immunoglobulin (Ig) heavy chain constant region or its fragment that retains the ability to CC CC bind an Fc receptor linked by a linker group or a direct bond to a CC peptide capable of binding an amyloidogenic protein. The invention is CC useful for clearing an amyloidogenic protein such as beta-amyloid, transthyretin (TTR), prion protein (PrP), islet amyloid polypeptide CC CC (IAPP), atrial natriuretic factor (ANF), kappa light chain, lambda light chain, amyloid A, procalcitonin, cystatin C, beta2-microglobulin, ApoA-I, CC CC gelsolin, calcitonin, fibrinogen, Huntington, alpha-synuclein and lysozyme from a subject and for treating an amyloidogenic disorder such CC as Alzheimer's disease and spongiform encephalopathy. Disorders treatable CCinclude those caused or characterised by deposits of TTR (eg. familial CCamyloid cardiomyopathy), PrP (eg. spongiform encephalopathies, including CC scrapie in sheep, bovine spongiform encephalopathy in cows and CC Creutzfeldt-Jacob disease (CJ) and Gerstmann-Straussler-Scheinker CC syndrome (GSS) in humans), IAPP (eq. insulinoma, adult onset diabetes), CC ANF (eq. isolated atrial amyloid), kappa or lambda light chain (eg. CC idiopathic amyloidosis, myeloma), amyloid A (eq. amyloidosis), Apo A-I CC (eq. hereditary non-neuropathic systemic amyloidosis), Gelsolin (eq. CCCCfamilial amyloidosis of Finnish type), Fibrinogen (eg. hereditary renal

amyloidosis), Lysozyme (eg. hereditary systemic amyloidosis). Other

examples of amyloidogenic disorders include Huntington's disease and inclusion body myocytis. The present sequence is human beta-amyloid

CC

CC

```
CC
     peptide mutant. Note: This sequence is not shown in the specification but
     is derived from human beta-amyloid peptide shown as SEQ ID NO: 1
CC
CC
     (AAE26265) in the specification
XX
SO
     Sequence 16 AA;
  Query Match
                          85.4%; Score 35; DB 5; Length 16;
                          100.0%; Pred. No. 2.4;
  Best Local Similarity
                                                                              0;
 Matches
            7; Conservative
                                 0; Mismatches
                                                   0; Indels
                                                                  0; Gaps
            1 LVFFAED 7
Qу
              8 LVFFAED 14
Db
RESULT 52
AAR54703
     AAR54703 standard; peptide; 17 AA.
XX
AC
     AAR54703;
XX
DT
     25-MAR-2003
                  (revised)
DΤ
     15-DEC-1994
                 (first entry)
XX
DE
     Beta-amyloid fragment (12-28).
XX
KW
     Beta-amyloid protein; BAP; Alzheimer's disease; diagnosis.
XX
OS
     Homo sapiens.
XX
PN
     WO9409364-A1.
XX
PD
     28-APR-1994.
XX
PF
     13-OCT-1993;
                    93WO-US009772.
XX
PR
     13-OCT-1992;
                    92US-00959251.
XX
     (UYDU-) UNIV DUKE.
PA
XX
PΙ
     Strittmatter WJ;
XX
     WPI; 1994-151484/18.
DR
XX
     Immobilised beta-amyloid protein or fragments - used in assays for
PΤ
     obtaining prods for use in the diagnosis and treatment of disorders such
PT
     as Alzheimer's disease.
PT
XX
PS
     Claim 5; Page 28; 49pp; English.
XX
     A construct comprising a beta-amyloid protein (BAP) or fragment (esp. the
CC
     peptides given in AAR54702-03) immobilised on a solid support can be used
CC
     to detect cpds. which bind to BAP. Binding of proteins in human
CC
     cerebrospinal fluid proteins were shown to bind to beta- amyloid peptides
CC
     1-28 and 12-28. Hydropathic mimic peptide (12-28) was used as control.
CC
CC
     (Updated on 25-MAR-2003 to correct PN field.)
XX
```

```
Sequence 17 AA;
SO
                          85.4%; Score 35; DB 2; Length 17;
  Query Match
  Best Local Similarity
                          100.0%; Pred. No. 2.5;
  Matches
            7; Conservative
                                0; Mismatches
                                                    0; Indels
                                                                  0; Gaps
                                                                              0;
            1 LVFFAED 7
Qу
              111111
            6 LVFFAED 12
Db
RESULT 53
AAW18880
     AAW18880 standard; peptide; 17 AA.
XX
AC
     AAW18880;
XX
DT
     08-DEC-1997
                  (first entry)
XX
DΕ
     Beta-amyloid peptide fragment (9-25).
XX
     beta-amyloid peptide; membrane protein; amyloid precursor protein;
ΚW
     fibril assembly; in vitro; detection; fluorescence; amyloidosis disorder;
KW
     Alzheimer's disease; multiple myeloma; rheumatoid arthritis; diabetes;
KW
ΚW
     prion disorder.
XX
OS
     Synthetic.
XX
PN
     WO9707402-A1.
XX
PD
     27-FEB-1997.
XX
                    96WO-CA000555.
PF
     16-AUG-1996;
XX
PR
     17-AUG-1995;
                    95US-00515615.
XX
PΑ
     (ONTA-) ONTARIO CANCER INST.
XX
PΙ
     Chakrabartty A;
XX
DR
     WPI; 1997-165446/15.
XX
     In vitro fluorescence monitoring of protein fibril assembly - esp. useful
PT
PT
     for monitoring fibril assembly processes associated with amyloidosis
PT
     disorders, esp. Alzheimer's disease.
XX
PS
     Disclosure; Page 24; 40pp; English.
XX
     This peptide is a fibrillogenic fragment of beta-amyloid peptide (a
CC
     fragment of the integral membrane protein, amyloid precursor protein).
CC
     Beta-amyloid protein fibril assembly can be monitored using a new method
CC
     for in vitro monitoring of peptide/protein fibril assembly using
CC
     fluorescent energy transfer between closely juxtaposed donor and acceptor
CC
     fluorophores. Two forms of beta-amyloid (9-25) were synthesised, one had
CC
CC
     a Trp residue attached to the N-terminus of the peptide (AAW18881), and
CC
     the other (AAW18882) had a cysteine residue attached to the N-terminus,
```

and an AEDANS group chemically linked to the sulfhydryl side chain of the

CC

```
CC
     cysteine. When both forms of beta-amyloid are mixed together, fibrils
    will assemble and in the fibril state the Trp and AEDANS groups will be
CC
CC
     closer in space than in the non-fibril state. Fluorescence energy
CC
     transfer between Trp and AEDANS increases when the two fluorphores are
CC
     close in space (i.e. efficiency of energy transfer will increase as the
CC
     fibrils form) and the fluorescence can be measured. Fibril assembly
CC
    processes associated with various amyloidosis disorders can be monitored
    by the method, especially Alzheimer's disease (claimed), multiple
CC
    myeloma, rheumatoid arthritis, diabetes and prion disorders
CC
XX
SQ
     Sequence 17 AA;
                          85.4%; Score 35; DB 2; Length 17;
  Query Match
                          100.0%; Pred. No. 2.5;
  Best Local Similarity
                              0; Mismatches
 Matches
            7; Conservative
                                                   0; Indels
                                                                 0; Gaps
                                                                             0;
            1 LVFFAED 7
Qy
             Db
            9 LVFFAED 15
RESULT 54
AAB91774
    AAB91774 standard; peptide; 17 AA.
XX
AC
    AAB91774;
XX
    22-JUN-2001 (first entry)
DT
XX
    Amyloid beta-protein fragment peptide SEQ ID NO:950.
DE
XX
     Protection; endogenous therapeutic peptide; peptidase; conjugation;
KW
KW
     blood component; modification; succinimidyl; maleimido group; amino;
     hydroxyl; thiol; hormone; growth factor; neurotransmitter.
KW
XX
OS
    Homo sapiens.
OS
     Synthetic.
XX
    WO200069900-A2.
PN
XX
PD
     23-NOV-2000.
XX
     17-MAY-2000; 2000WO-US013576.
PF
XX
PR
     17-MAY-1999;
                    99US-0134406P.
PR
     10-SEP-1999;
                    99US-0153406P.
     15-OCT-1999;
                    99US-0159783P.
PR
XX
PA
     (CONJ-) CONJUCHEM INC.
XX
     Bridon DP, Ezrin AM, Milner PG, Holmes DL, Thibaudeau K;
PΙ
XX
DR
     WPI; 2001-112059/12.
XX
     Modifying and attaching therapeutic peptides to albumin prevents
PT
PT
     peptidase degradation, useful for increasing length of in vivo activity.
XX
```

```
PS
    Disclosure; Page 504; 733pp; English.
XX
CC
     The present invention describes a modified therapeutic peptide (I)
CC
     comprising a therapeutically active amino acid region (III) and a
CC
     reactive group (II) (e.g. succinimidyl and maleimido groups) attached to
CC
     a less therapeutically active amino acid region (IV), which covalently
CC
     bonds with amino/hydroxyl/thiol groups on blood components to form a
     peptidase stabilised therapeutic peptide composed of 3-50 amino acids.
CC
     (I) are useful for modifying therapeutic peptides e.g. hormones, growth
CC
CC
     factors and neurotransmitters, to protect them from peptidase activity in
CC
     vivo for the treatment of various disorders. Endogenous therapeutic
    peptides are not suitable as drug candidates as they require frequent
CC
     administration due to rapid degradation by peptidases in the body.
CC
CC
    Modifying and attaching therapeutic peptides to albumin prevents or
CC
     reduces the action of peptidases to increase length of activity (half
     life) and specificity as bonding to large molecules decreases
CC
     intracellular uptake and interference with physiological processes.
CC
CC
     AAB90829 to AAB92441 represent peptides which can be used in the
CC
     exemplification of the present invention
XX
SQ
     Sequence 17 AA;
  Query Match
                          85.4%; Score 35; DB 4; Length 17;
  Best Local Similarity
                          100.0%; Pred. No. 2.5;
                                                                             0;
  Matches
            7; Conservative
                              0; Mismatches
                                                   0; Indels
                                                                 0; Gaps
Qy
            1 LVFFAED 7
              6 LVFFAED 12
Db
RESULT 55
AAB91807
ID
    AAB91807 standard; peptide; 17 AA.
XX
AC
    AAB91807;
XX
DT
     22-JUN-2001 (first entry)
XX
DE
     Amyloid beta-protein fragment peptide SEQ ID NO:983.
XX
KW
     Protection; endogenous therapeutic peptide; peptidase; conjugation;
     blood component; modification; succinimidyl; maleimido group; amino;
KW
KW
     hydroxyl; thiol; hormone; growth factor; neurotransmitter.
XX
OS
     Homo sapiens.
OS
     Synthetic.
XX
PN
     WO200069900-A2.
XX
PD
     23-NOV-2000.
XX
     17-MAY-2000; 2000WO-US013576.
PF
XX
PR
     17-MAY-1999;
                    99US-0134406P.
PR
     10-SEP-1999;
                    99US-0153406P.
PR
     15-OCT-1999;
                    99US-0159783P.
```

```
XX
PΑ
     (CONJ-) CONJUCHEM INC.
XX
PΙ
     Bridon DP, Ezrin AM, Milner PG, Holmes DL, Thibaudeau K;
XX
DR
    WPI; 2001-112059/12.
XX
    Modifying and attaching therapeutic peptides to albumin prevents
PT
    peptidase degradation, useful for increasing length of in vivo activity.
PT
XX
PS
    Disclosure; Page 516; 733pp; English.
XX
    The present invention describes a modified therapeutic peptide (I)
CC
CC
     comprising a therapeutically active amino acid region (III) and a
CC
     reactive group (II) (e.g. succinimidyl and maleimido groups) attached to
     a less therapeutically active amino acid region (IV), which covalently
CC
    bonds with amino/hydroxyl/thiol groups on blood components to form a
CC
    peptidase stabilised therapeutic peptide composed of 3-50 amino acids.
CC
CC
     (I) are useful for modifying therapeutic peptides e.g. hormones, growth
     factors and neurotransmitters, to protect them from peptidase activity in
CC
     vivo for the treatment of various disorders. Endogenous therapeutic
CC
     peptides are not suitable as drug candidates as they require frequent
CC
     administration due to rapid degradation by peptidases in the body.
CC
    Modifying and attaching therapeutic peptides to albumin prevents or
CC
     reduces the action of peptidases to increase length of activity (half
CC
     life) and specificity as bonding to large molecules decreases
CC
     intracellular uptake and interference with physiological processes.
CC
CC
    AAB90829 to AAB92441 represent peptides which can be used in the
CC
     exemplification of the present invention
XX
SO
     Sequence 17 AA;
                          85.4%; Score 35; DB 4; Length 17;
  Query Match
                          100.0%; Pred. No. 2.5;
  Best Local Similarity
                                 0; Mismatches
                                                   0;
                                                                     Gaps
                                                                             0;
 Matches
            7; Conservative
                                                       Indels
                                                                 0;
            1 LVFFAED 7
Qу
              Db
            6 LVFFAED 12
RESULT 56
AAB48346
    AAB48346 standard; peptide; 17 AA.
XX
AC
    AAB48346;
XX
DT
     20-APR-2001 (first entry)
XX
     Beta-amyloid antigenic peptide (Abeta10-25).
DΕ
XX
KW
     Beta-amyloid; nootropic; neuroprotective; vaccine; antibody; brain;
     amyloid plaque; Alzheimer's disease; antigen.
KW
XX
OS
     Homo sapiens.
XX
                    Location/Qualifiers
FΗ
     Key
```

```
FT
    Modified-site
                     17
FT
                     /note= "C-terminal amide"
XX
PN
    WO200077178-A1.
XX
PD
     21-DEC-2000.
XX
ΡF
     15-JUN-2000; 2000WO-US016551.
XX
PR
     16-JUN-1999;
                    99US-0139408P.
XX
PΑ
     (BOST-) BOSTON BIOMEDICAL RES INST.
XX
PΙ
    Raso V;
XX
DR
    WPI; 2001-112220/12.
XX
PT
     New antibodies which catalyze hydrolysis of beta-amyloid at a
PT
    predetermined amide linkage, useful for e.g. sequestering or reducing
PT
     free beta-amyloid in the bloodstream and brain and preventing formation
PT
     of amyloid plaques.
XX
PS
     Example 1; Fig 3; 82pp; English.
XX
     The invention relates to an antibody which catalyzes the hydrolysis of
CC
     beta-amyloid at a predetermined amide linkage. The antibodies are useful
CC
CC
     for sequestering free beta-amyloid in the bloodstream of an animal,
     reducing beta-amyloid levels in the brain, preventing formation of
CC
     amyloid plaques, and disaggregating amyloid plaques present in the brain,
CC
CC
     thus may be used in treating patients diagnosed with or at risk for
CC
     Alzheimer's disease. The present sequence represents a beta-amyloid
CC
     antigenic peptide made from the central region of beta-amyloid. The
CC
     antigenic peptides were designed to be tested for suitability to antibody
CC
     -mediated therapy
XX
     Sequence 17 AA;
SQ
  Query Match
                          85.4%; Score 35; DB 4; Length 17;
                          100.0%; Pred. No. 2.5;
  Best Local Similarity
             7; Conservative 0; Mismatches 0; Indels
                                                                 0; Gaps
            1 LVFFAED 7
Qу
              9 LVFFAED 15
RESULT 57
ABB04911
    ABB04911 standard; peptide; 17 AA.
TD
XX
AC
    ABB04911;
XX
     14-MAR-2002 (first entry)
DT
XX
DΕ
     Human amyloid beta protein (beta-A4) peptide 12-28 SEQ ID NO:2.
XX
KW
     Human; amyloid beta protein; beta-A4; memory enhancement; learning.
```

```
XX
OS
    Homo sapiens.
XX
PN
    US6320024-B1.
XX
PD
     20-NOV-2001.
XX
PF
     09-MAR-1999;
                    99US-00264709.
XX
PR
     07-FEB-1997;
                    97US-00797782.
XX
PΑ
     (ROBE/) ROBERTS E.
XX
PI
    Roberts E;
XX
DR
    WPI; 2002-096566/13.
XX
PT
    New peptide compound useful for design of substances that enhance memory.
XX
PS
     Disclosure; Col 1; 30pp; English.
XX
CC
     The present invention describes a novel peptide compound comprising Lys-
CC
    His-Tyr-beta-alanine, which has a memory modulating effect. The peptide
    has nootropic activity. The peptide can be used for the development of
CC
     topographic models useful to design and synthesise memory-enhancing and
CC
CC
    life-quality improving substances. The peptide compound restores the
CC
    balance between excitatory and inhibitory systems in the brain, which is
     required for optimal acquisition and retention of learning and helps to
CC
     correct defects in the balance that arise as a result of aging,
CC
CC
     infections and injury. The substances exert recyberneticising effects on
CC
    nervous system function and has more prolonged desired effects at lower
CC
    doses than the peptide structures. The substances mimic the action of
     active peptides without having a peptide structure and do not subject to
CC
    degradation of peptide-splitting enzymes in the gut or other tissues. The
CC
    present sequence represents a human amyloid beta protein (beta-A4)
CC
CC
    peptide, which is used in the exemplification of the present invention
XX
SQ
     Sequence 17 AA;
                          85.4%; Score 35; DB 5; Length 17;
  Query Match
  Best Local Similarity
                          100.0%; Pred. No. 2.5;
             7; Conservative
                               0; Mismatches
                                                                  0; Gaps
                                                                              0;
 Matches
                                                    0; Indels
            1 LVFFAED 7
Qу
              111111
            6 LVFFAED 12
Db
RESULT 58
ABB99611
ID
    ABB99611 standard; peptide; 17 AA.
XX
AC
    ABB99611;
XX
DT
     28-MAR<sup>≜</sup>2003 (first entry)
XX
DΕ
     Peptide derived from human amyloid precursor protein (APP).
```

```
XX
KW
     Amyloid precursor protein; APP; protein derivative;
KW
     neurodegenerative disease; Alzheimer's disease; cognitive enhancer.
XX
OS
     Synthetic.
OS
     Homo sapiens.
XX
ΡN
     WO200283729-A2.
XX
PD
     24-OCT-2002.
XX
PF
     17-APR-2002; 2002WO-GB001769.
XX
PR
     18-APR-2001; 2001GB-00009558.
PR
     17-AUG-2001; 2001GB-00020084.
PR
     30-NOV-2001; 2001US-00998491.
PR
     28-MAR-2002; 2002GB-00007387.
XX
PA
     (UYOP-) UNIV OPEN.
XX
PI
     Mileusnic R, Rose SPR;
XX
DR
     WPI; 2003-111814/10.
XX
     Derivatives of polypeptides, useful for treating neurodegenerative
PT
PT
     disease e.g. Alzheimer's disease, comprises one functional amino acid
PT
     residue or derivative protected by a protective group.
XX
PS
     Disclosure; Page 3; 85pp; English.
XX
CC
     The present sequence is derived from amyloid precursor protein (APP).
CC
     Derivatives of the invention are based on APP sequences. The
     specification describes a derivative of a polypeptide in which at least
CC
     one functional group of at least one amino acid residue or derivative is
CC
     protected by a protective group. This derivative is of the formula given
CC
CC
     in ABB99625. The derivative is useful in medicine and in the preparation
CC
     of a medicament for use in the treatment of a neurodegenerative disease
CC
     e.g. Alzheimer's disease. It is also useful as a cognitive enhancer
XX
     Sequence 17 AA;
SQ
                          85.4%; Score 35; DB 6; Length 17;
  Query Match
                          100.0%; Pred. No. 2.5;
  Best Local Similarity
            7; Conservative
                               0; Mismatches
                                                   0; Indels
                                                                  0; Gaps
                                                                              0;
            1 LVFFAED 7
Qу
              1111111
            6 LVFFAED 12
RESULT 59
AAB10963
     AAB10963 standard; protein; 18 AA.
ID
XX
AC
    AAB10963;
XX
DT
     07-FEB-2001 (first entry)
```

XX DE Beta-amyloid precursor protein peptide fragment. XX KW APP; amyloid precursor protein; human; alpha-secretase; ADAM 10; ΚW disintegrin-metalloprotease; protease; nootropic; neuroprotective; KW gene therapy; Alzheimer's disease. XX os Unidentified. XX PNDE19910108-A1. XX PD 21-SEP-2000. XX PF08-MAR-1999; 99DE-01010108. XX PR 08-MAR-1999; 99DE-01010108. XX PΑ (FAHR/) FAHRENHOLZ F.

XX

PI Fahrenholz F, Postina R;

WPI; 2000-588391/56.

DR

XX PT

PT

PT

XX PS

XX CC

XX

Recombinant cells, for identifying alpha-secretase active agents and identifying risk factors associated with Alzheimer's disease, comprise amyloid precursor protein and alpha-secretase.

Example 13; Page 12; 24pp; German.

This invention describes a novel recombinant cell comprising recombinant nucleic acids encoding a region of human amyloid precursor protein containing an alpha-secretase cleavage site and a protease or a heterologous RNA coding for a substrate protein and a protease. The invention also describes a recombinant cell, characterized in that it contains recombinant nucleic acids comprising either: (a) a gene for a substrate protein (SP), which comprises a sequence region of 18 amino acids of the human amyloid precursor protein (APP) or a homologous protein, where the sequence region contains the alpha-secretase cleavage site at a reference of 6 residues at the N-terminal and 12 residues at the C-terminal; and (b) a gene for a protease protein (PP), that either comprises a proteolytically active necessary sequence region or a sequence region of the disintegrin metalloprotease ADAM 10 from a cow (Bos taurus), from a human or other mammal or a mutant of this, which shows the same enzymatic properties, where the genes are under the control of heterologous promoters; or a heterologous RNA coding for a SP and a PP. The products of the invention have nootropic and neuroprotective activity and can be used for gene therapy. The protease proteins of the invention are useful for proteolytic cleavage of substrate proteins, especially human amyloid precursor protein. Dominant negative forms of bovine, human or other mammalian disintegrinmetalloprotease ADAM 10 proteins and their coding sequences are useful for suppressing the alpha-secretase activity of a cell. Nucleic acid sequences encoding the proteases are useful for constructing vectors for gene therapy. The proteins and recombinant cells are useful for identifying secretases and pharmaceutical agents and to identify risk factors associated with Alzheimer's disease

```
Sequence 18 AA;
SO
  Query Match
                          85.4%; Score 35; DB 3;
                                                    Length 18;
  Best Local Similarity
                          100.0%; Pred. No. 2.7;
 Matches
             7; Conservative
                                0; Mismatches
                                                   0; Indels
                                                                  0; Gaps
                                                                              0;
Qу
            1 LVFFAED 7
              7 LVFFAED 13
Db
RESULT 60
AAW18882
ΙD
    AAW18882 standard; peptide; 19 AA.
XX
AC
    AAW18882;
XX
DT
     08-DEC-1997 (first entry)
XX
DΕ
    AEDANS-beta-amyloid peptide fragment (9-25).
XX
KW
     beta-amyloid peptide; membrane protein; amyloid precursor protein;
KW
     fibril assembly; in vitro; detection; fluorescence; amyloidosis disorder;
     Alzheimer's disease; multiple myeloma; rheumatoid arthritis; diabetes;
KW
KW
    prion disorder.
XX
OS
     Synthetic.
XX
FH
     Key
                     Location/Qualifiers
    Modified-site
FT
FT
                     /note= "AEDANS-Ac-Cys"
FT
     Modified-site
                     /note= "Gly-CONH2"
FT
XX
РΝ
     WO9707402-A1.
XX
PD
     27-FEB-1997.
XX
PF
     16-AUG-1996;
                    96WO-CA000555.
XX
PR
     17-AUG-1995;
                    95US-00515615.
XX
     (ONTA-) ONTARIO CANCER INST.
PA
XX
     Chakrabartty A;
PI
XX
     WPI; 1997-165446/15.
DR
XX
PT
     In vitro fluorescence monitoring of protein fibril assembly - esp. useful
     for monitoring fibril assembly processes associated with amyloidosis
PT
PT
     disorders, esp. Alzheimer's disease.
XX
PS
     Claim 26; Page 25; 40pp; English.
XX
CC
     Beta-amyloid protein fibril assembly can be monitored using a new method
CC
     for in vitro monitoring of peptide/protein fibril assembly using
CC
     fluorescent energy transfer between closely juxtaposed donor and acceptor
```

```
fluorophores. Two forms of beta-amyloid (9-25) were synthesised, one had
CC
CC
     a Trp residue attached to the N-terminus of the peptide (AAW18881), and
CC
     the other (AAW18882) had a cysteine residue attached to the N-terminus,
     and an AEDANS group chemically linked to the sulfhydryl side chain of the
CC
     cysteine. When both forms of beta-amyloid are mixed together, fibrils
CC
CC
    will assemble and in the fibril state the Trp and AEDANS groups will be
CC
     closer in space than in the non-fibril state. Fluorescence energy
CC
     transfer between Trp and AEDANS increases when the two fluorphores are
CC
     close in space (i.e. efficiency of energy transfer will increase as the
CC
     fibrils form) and the fluorescence can be measured. Fibril assembly
CC
    processes associated with various amyloidosis disorders can be monitored
CC
    by the method, especially Alzheimer's disease (claimed), multiple
    myeloma, rheumatoid arthritis, diabetes and prion disorders
CC
XX
SQ
     Sequence 19 AA;
                          85.4%; Score 35; DB 2; Length 19;
  Query Match
  Best Local Similarity
                          100.0%; Pred. No. 2.8;
  Matches
            7; Conservative
                               0; Mismatches
                                                   0; Indels
                                                                 0; Gaps
                                                                              0;
           1 LVFFAED 7
Qy
              Db
           11 LVFFAED 17
RESULT 61
AAW18881
    AAW18881 standard; peptide; 19 AA.
XX
AC
    AAW18881;
XX
DT
     08-DEC-1997 (first entry)
XX
     Trp-Beta-amyloid peptide fragment (9-25).
DE
XX
     beta-amyloid peptide; membrane protein; amyloid precursor protein;
KW
     fibril assembly; in vitro; detection; fluorescence; amyloidosis disorder;
KW
     Alzheimer's disease; multiple myeloma; rheumatoid arthritis; diabetes;
KW
ΚW
     prion disorder.
XX
OS
     Synthetic.
XX
FH
                     Location/Qualifiers
FT
     Modified-site
                     /note= "Acetyl-Trp"
FT
     Modified-site
FT
                     /note= "Gly-CONH2"
FT
XX
     WO9707402-A1.
PN
XX
     27-FEB-1997.
PD
XX
PF
     16-AUG-1996;
                    96WO-CA000555.
XX
                    95US-00515615.
PR
     17-AUG-1995;
XX
PA
     (ONTA-) ONTARIO CANCER INST.
```

```
XX
PΙ
     Chakrabartty A;
XX
DR
     WPI; 1997-165446/15.
XX
PT
     In vitro fluorescence monitoring of protein fibril assembly - esp. useful
     for monitoring fibril assembly processes associated with amyloidosis
PT
     disorders, esp. Alzheimer's disease.
PT
XX
PS
     Claim 36; Page 25; 40pp; English.
XX
     Beta-amyloid protein fibril assembly can be monitored using a new method
CC
     for in vitro monitoring of peptide/protein fibril assembly using
CC
     fluorescent energy transfer between closely juxtaposed donor and acceptor
CC
     fluorophores. Two forms of beta-amyloid (9-25) were synthesised, one had
CC
     a Trp residue attached to the N-terminus of the peptide (AAW18881), and
CC
     the other (AAW18882) had a cysteine residue attached to the N-terminus,
CC
     and an AEDANS group chemically linked to the sulfhydryl side chain of the
CC
CC
     cysteine. When both forms of beta-amyloid are mixed together, fibrils
     will assemble and in the fibril state the Trp and AEDANS groups will be
CC
     closer in space than in the non-fibril state. Fluorescence energy
CC
     transfer between Trp and AEDANS increases when the two fluorphores are
CC
     close in space (i.e. efficiency of energy transfer will increase as the
CC
     fibrils form) and the fluorescence can be measured. Fibril assembly
CC
     processes associated with various amyloidosis disorders can be monitored
CC
     by the method, especially Alzheimer's disease (claimed), multiple
CC
     myeloma, rheumatoid arthritis, diabetes and prion disorders
CC
XX
     Sequence 19 AA;
SQ
  Query Match
                          85.4%; Score 35; DB 2; Length 19;
                                  Pred. No. 2.8;
  Best Local Similarity
                          100.0%;
  Matches
             7; Conservative
                                0; Mismatches
                                                   0; Indels
                                                                  0; Gaps
                                                                              0;
            1 LVFFAED 7
Qу
              111111
Db
           11 LVFFAED 17
RESULT 62
AAY79935
     AAY79935 standard; peptide; 19 AA.
ID
XX
AC
     AAY79935;
XX
DT
     11-MAY-2000 (first entry)
XX
DE
     Beta-amyloid inhibitor peptide SEQ ID NO:11.
XX
     Beta-amyloid; inhibitor; recognition element; hybrid; aggregation;
KW
     Alzheimer's disease; neuroprotective; nootropic.
KW
XX
OS
     Homo sapiens.
OS
     Synthetic.
XX
PN
     US6022859-A.
XX
```

```
PD
    08-FEB-2000.
XX
PF
     14-NOV-1997;
                   97US-00970833.
XX
PR
     15-NOV-1996;
                   96US-0030840P.
XX
PΑ
     (WISC ) WISCONSIN ALUMNI RES FOUND.
XX
PΙ
    Murphy RM,
                Kiessling LL;
XX
DR
    WPI; 2000-160387/14.
XX
PT
    Beta-amyloid inhibitor useful for treating Alzheimer's disease.
XX
PS
    Claim 3; Col 19-20; 15pp; English.
XX
    The present sequence represents a beta-amyloid inhibitor peptide. Beta-
CC
CC
     amyloid inhibitors have neuroprotective and nootropic properties. The
CC
     inhibitor peptides are useful for the treatment of Alzheimer's disease
XX
    Sequence 19 AA;
SQ
  Query Match
                          85.4%; Score 35; DB 3; Length 19;
                         100.0%; Pred. No. 2.8;
  Best Local Similarity
            7; Conservative 0; Mismatches
                                                                             0;
 Matches
                                                   0; Indels
                                                                 0; Gaps
Qу
            1 LVFFAED 7
              11 LVFFAED 17
Db
RESULT 63
AAB49097
ID
    AAB49097 standard; peptide; 19 AA.
XX
AC
    AAB49097;
XX
DT
    27-MAR-2001 (first entry)
XX
DΕ
    Human amyloid beta peptide (residues 13-28), SEQ ID NO:33.
XX
    Amyloid disease; amyloid fibril deposition; amyloid plaque; immunogenic;
KW
     antibody; vaccine; Alzheimer's disease; type 2 diabetes;
KW
     reactive system amyloidosis; systemic senile amyloidosis;
KW
     familial amyloid cardiomyopathy; transmissible spongiform encephalopathy;
KW
     Creutzfeld-Jakob disease; Kuru;
KW
     haemodialysis-asssociated beta-2-microglobulin deposition;
KW
KW
     amyloid beta peptide.
XX
OS
     Homo sapiens.
XX
PN
     WO200072876-A2.
XX
     07-DEC-2000.
PD
XX
PF
     01-JUN-2000; 2000WO-US015239.
XX
```

```
01-JUN-1999;
                    99US-0137010P.
PR
XX
PΑ
     (NEUR-) NEURALAB LTD.
XX
PΙ
     Schenk DB;
XX
DR
     WPI; 2001-070921/08.
XX
     Pharmaceutical composition comprising immunogen against amyloid component
PT
PT
     such as fibril peptide or protein, or antibody against amyloid component
PT
     useful for treating amyloid diseases or amyloidoses.
XX
PS
     Example IV; Page 74; 140pp; English.
XX
CC
     The invention relates to a novel pharmaceutical composition for
CC
     preventing or treating a disease characterised by amyloid fibril deposits
     (amyloid plaques) in a patient. The pharmaceutical composition comprises
CC
CC
     an agent that will induce an immune response against an amyloid
CC
     component, or an antibody or antibody fragment that binds to an amyloid
CC
     component. The invention also relates to a method for determining the
CC
     prognosis of a patient undergoing treatment for an amyloid disorder which
     involves measuring a patient serum amount of immunoreactivity against a
CC
CC
     selected amyloid component. A patient serum immunoreactivity of at least
CC
     four times a base line serum immunoreactivity control level indicates a
     prognosis of improved status with respect to the disorder. The
CC
CC
     pharmaceutical compositions of the invention are useful for treating a
CC
     wide variety of disorders characterised by amyloid fibril deposition in a
     patient. Such disorders include Alzheimer's disease characterised by
CC
     amyloid beta peptide fibril deposits; type 2 diabetes characterised by
CC
     islet amyloid protein peptide (IAPP, amylin) fibrils; reactive systemic
CC
CC
     amyloidosis associated with systemic inflammatory diseases (e.g.,
     rheumatoid arthritis, osteomyelitis, tuberculosis) characterised by AA
CC
     fibrils derived from serum amyloid A protein (ApoSSA)); systemic senile
CC
     amyloidosis and familial amyloid cardiomyopathy characterised by ATTR
CC
     fibrils derived from transthyretin (TTR); transmissible spongiform
CC
CC
     encephalopathies (e.g. Creutzfeld-Jakob disease, Kuru) characterised by
     prion protein deposits; and beta-2-microglobulin deposits which form as a
CC
     result of long term haemodialysis treatment. The present sequence
CC
CC
     represents a human amyloid beta peptide which was conjugated to sheep
CC
     anti-mouse IgG in an exemplification of the invention
XX
SQ
     Sequence 19 AA;
                          85.4%; Score 35; DB 4; Length 19;
  Query Match
                          100.0%; Pred. No. 2.8;
  Best Local Similarity
                                0; Mismatches
                                                   0; Indels
                                                                             0;
  Matches
             7; Conservative
                                                                 0;
                                                                     Gaps
            1 LVFFAED 7
Qу
              5 LVFFAED 11
Db
RESULT 64
AAB46201
     AAB46201 standard; peptide; 19 AA.
ID
XX
AC
     AAB46201;
```

```
XX
DT
     04-APR-2001 (first entry)
XX
DE
     Human APP A-beta 13-28 peptide.
XX
KW
     Amyloid deposit; APP; Abeta; brain; human; clearing response; nootropic;
KW
     Fc receptor mediated phagocytosis; immunogenic response; neuroprotective;
KW
     amyloid precursor protein; Alzheimer's disease.
XX
OS
     Homo sapiens.
XX
     WO200072880-A2.
PN
XX
     07-DEC-2000.
PD
XX
PF
     26-MAY-2000; 2000WO-US014810.
XX
     28-MAY-1999;
                    99US-00322289.
PR
XX
PA
     (NEUR-) NEURALAB LTD.
XX
PΙ
     Schenk DB, Bard F, Vasquez NJ, Yednock T;
XX
DR
     WPI; 2001-032104/04.
XX
PT
     Preventing or treating a disease associated with amyloid deposits,
PT
     especially Alzheimer's disease, comprises administering amyloid specific
PT
     antibody.
XX
PS
     Disclosure; Page 61; 143pp; English.
XX
CC
     This invention describes a novel method of preventing or treating a
     disease associated with amyloid deposits of amyloid precursor protein
CC
     (APP) Abeta fragments in the brain of a patient, which comprises
CC
CC
     administering to the patient: (a) an antibody that binds to Abeta, the
CC
     antibody binds to an amyloid deposit and induces a clearing response (Fc
CC
     receptor mediated phagocytosis) against it (b) a polypeptide containing
CC
     an N-terminal segment of at least residues 1-5 of Abeta; or (c) an agent
CC
     that induces an immunogenic response against residues 1-3 to 7-11 of
CC
     Abeta. The products of the invention have nootropic and neuroprotective
     activity. The method is also useful for monitoring a course of treatment
CC
CC
     being administered to a patient e.g. active and passive immunization. The
CC
     methods are useful for prophylactic and therapeutic treatment of
CC
     Alzheimer's disease
XX
     Sequence 19 AA;
SO
  Query Match
                          85.4%; Score 35; DB 4; Length 19;
                          100.0%; Pred. No. 2.8;
  Best Local Similarity
            7; Conservative
                                0; Mismatches
                                                   0;
                                                      Indels
                                                                     Gaps
                                                                             0;
Qу
            1 LVFFAED 7
              Db
            5 LVFFAED 11
```

```
AAY79934
ID
     AAY79934 standard; peptide; 20 AA.
XX
AC
     AAY79934;
XX
DT
     11-MAY-2000 (first entry)
XX
DE
     Beta-amyloid inhibitor peptide SEQ ID NO:10.
XX
KW
     Beta-amyloid; inhibitor; recognition element; hybrid; aggregation;
KW
     Alzheimer's disease; neuroprotective; nootropic.
XX
OS
     Homo sapiens.
OS
     Synthetic.
XX
ΡN
     US6022859-A.
XX
PD
     08-FEB-2000.
XX
PF
     14-NOV-1997;
                    97US-00970833.
XX
PR
     15-NOV-1996;
                    96US-0030840P.
XX
PΑ
     (WISC ) WISCONSIN ALUMNI RES FOUND.
XX
PI
     Murphy RM,
                Kiessling LL;
XX
     WPI; 2000-160387/14.
DR
XX
PT
     Beta-amyloid inhibitor useful for treating Alzheimer's disease.
XX
PS
     Claim 2; Col 17-18; 15pp; English.
XX
CC
     The present sequence represents a beta-amyloid inhibitor peptide. Beta-
     amyloid inhibitors have neuroprotective and nootropic properties. The
CC
CC
     inhibitor peptides are useful for the treatment of Alzheimer's disease
XX
SQ
     Sequence 20 AA;
                          85.4%; Score 35; DB 3; Length 20;
  Query Match
  Best Local Similarity
                          100.0%; Pred. No. 3;
            7; Conservative 0; Mismatches
                                                                              0;
 Matches
                                                   0; Indels
                                                                  0; Gaps
            1 LVFFAED 7
Qу
              1111111
            4 LVFFAED 10
Db
RESULT 66
AAY30941
     AAY30941 standard; peptide; 21 AA.
XX
AC
     AAY30941;
XX
DT
     19-OCT-1999 (first entry)
XX
DΕ
     Human secretase SEC-alphal peptide fragment.
```

```
XX
KW
     Secretase; hyperforin; treatment; Alzheimer's disease; purification;
KW
     adhyperforin; St. John's Wort; storage stabile; pharmaceutical; symptom;
KW
     SEC-alpha1; human.
XX
OS
     Homo sapiens.
XX
PN
     WO9941220-A1.
XX
PD
     19-AUG-1999.
XX
PF
     04-FEB-1999;
                    99WO-EP000737.
XX
PR
     13-FEB-1998;
                    98DE-01005947.
XX
PA
     (SCHW-) SCHWABE GMBH & CO WILLMAR.
XX
PI
     Chatterjee SS,
                    Erdelmeier C, Klessing K, Marme D, Schaechtele C;
XX
DR
     WPI; 1999-508609/42.
XX
PT
     Hyperforin and adhyperforin isolated from St. John's Wort for treatment
PT
     of Alzheimers.
XX
PS
     Example 34; Fig 1; 41pp; German.
XX
CC
     This invention describes novel hyperforin and adhyperforin salts of
CC
     formula (I): (A-)m (B)p+, where m = 1-3; (A-) = an anion of formula (II);
CC
     n = 0-1; (B)p+ = an alkali metal ion or an ammonium ion of a salt-forming
CC
     nitrogen base of formula (III); R1-R3 = H, an optionally branched alkyl,
CC
     cycloalkyl, bicycloalkyl, tricycloalkyl, alkenyl, alkinyl,
     heterocycloalkyl, aryl, heteroaryl, arylalkyl or a heteroarylalkyl group,
CC
CC
     all optionally substituted with one or more hydroxy, alkoxy, aryloxy,
     alkanoyl, aroyl, carboxy, alkoxycarbamoyl, ureido, amidino, guanidino,
CC
CC
     cyano, azido, mercapto, alkylthio, alkylsulphoxy, alkylsulphonyl,
CC
     alkylsulphenyl, aminosulphonyl, fluoro, chloro, bromo, iodo, alkyl or
CC
     perfluoroalkyl; R1+R2 = together with an N-atom form, together with a N-
CC
    Atom an azetidin-, pyrrolidin-, pyrrolin-, piperidin-, piperazin-,
CC
     homopiperazin-, morpholin-, thiomorpholin-, pyridin-, di- or tetra-
CC
     hydropyridin-, pyrimidin-, pyrazin-, azepin-, dihydroazepin-, oxazepin-,
CC
     diazepin-, imidazol-, pyrazol-, oxazol- or thiazol-ring, optionally with
CC
     aliphatic, heteroaliphatic, aromatic or heteroaromatic rings or
CC
     substituted with hydroxy, alkoxy, aryloxy, alkanoyl, aroyl, carboxy,
CC
     alkoxycarbamoyl, ureido, amidino, quanidino, cyano, azido, mercapto,
     alkylthio, alkylsulphoxy, alkylsulphonyl, alkylsulphenyl, aminosulphonyl,
CC
CC
     fluoro, chloro, bromo, iodo, alkyl or perfluoroalkyl; R4 = H, or an
CC
     optionally branched alkyl group. The preparation is used to purify the
CC
     hyperforin and/or adhyperforin content in St. John's Wort extracts. The
CC
     obtained salts are storage stabile and can be used in pharmaceutical
     compositions for the treatment of Alzheimer's disease and its symptoms.
CC
CC
     This sequence represents a fragment of the human secretase SEC-alphal
CC
     protein which is used to illustrate the method of the invention
XX
SQ
     Sequence 21 AA;
  Query Match
                          85.4%; Score 35; DB 2; Length 21;
```

100.0%; Pred. No. 3.1;

Best Local Similarity

```
Matches
            7; Conservative 0; Mismatches
                                                   0; Indels
                                                                 0; Gaps
                                                                             0;
Qу
            1 LVFFAED 7
              Db
           12 LVFFAED 18
RESULT 67
AAR52569
     AAR52569 standard; peptide; 24 AA.
XX
AC
    AAR52569;
XX
DT
    16-DEC-1994 (first entry)
XX
DE
    Alzheimer's disease related immunogen.
XX
KW
     Alzheimer's disease; senile dementia; immunogen.
XX
OS
     Synthetic.
XX
PN
     JP06009693-A.
XX
PD
     18-JAN-1994.
XX
PF
     23-JAN-1992;
                    92JP-00031341.
XX
PR
     23-JAN-1992;
                    92JP-00031341.
XX
PΑ
     (EIKE ) EIKEN KAGAKU KK.
XX
DR
     WPI; 1994-146876/18.
XX
PT
     Alzheimer's disease related protein isolated from serum of patient -
PT-
     useful in diagnosis.
XX
PS
     Claim 1; Page 2; 8pp; Japanese.
XX
     A monoclonal antibody raised against the synthetic peptide AAR52569 as
CC
CC
     immunogen reacts with a new Alzheimer's disease related protein. The
     novel protein has a mol.wt. of 20kD (by SDS-PAGE), isoelectric point of
CC
CC
     ca. 5-7 and is abundant in serum of AD patients
XX
SQ
     Sequence 24 AA;
  Query Match
                          85.4%; Score 35; DB 2; Length 24;
                         100.0%; Pred. No. 3.6;
  Best Local Similarity
                                                   0; Indels
            7; Conservative 0; Mismatches
                                                                 0; Gaps
                                                                             0;
            1 LVFFAED 7
Qу
              17 LVFFAED 23
RESULT 68
AAB91832
     AAB91832 standard; peptide; 24 AA.
```

```
XX
AC
    AAB91832;
XX
    22-JUN-2001 (first entry)
DT
XX
DE
    Amyloid beta-protein fragment peptide SEQ ID NO:1008.
XX
KW
    Protection; endogenous therapeutic peptide; peptidase; conjugation;
KW
    blood component; modification; succinimidyl; maleimido group; amino;
KW
    hydroxyl; thiol; hormone; growth factor; neurotransmitter.
XX
OS
    Homo sapiens.
OS
    Synthetic.
XX
PN
    W0200069900-A2.
XX
    23-NOV-2000.
PD
XX
ΡF
    17-MAY-2000; 2000WO-US013576.
XX
PR
    17-MAY-1999;
                    99US-0134406P.
PR
    10-SEP-1999;
                    99US-0153406P.
PR
    15-OCT-1999;
                    99US-0159783P.
XX
PΑ
     (CONJ-) CONJUCHEM INC.
XX
PΙ
    Bridon DP, Ezrin AM, Milner PG, Holmes DL, Thibaudeau K;
XX
DR
    WPI; 2001-112059/12.
XX
PT
    Modifying and attaching therapeutic peptides to albumin prevents
PT
    peptidase degradation, useful for increasing length of in vivo activity.
XX
PS
    Disclosure; Page 525; 733pp; English.
XX
    The present invention describes a modified therapeutic peptide (I)
CC
CC
    comprising a therapeutically active amino acid region (III) and a
CC
     reactive group (II) (e.g. succinimidyl and maleimido groups) attached to
CC
    a less therapeutically active amino acid region (IV), which covalently
CC
    bonds with amino/hydroxyl/thiol groups on blood components to form a
CC
    peptidase stabilised therapeutic peptide composed of 3-50 amino acids.
CC
     (I) are useful for modifying therapeutic peptides e.g. hormones, growth
CC
     factors and neurotransmitters, to protect them from peptidase activity in
    vivo for the treatment of various disorders. Endogenous therapeutic
CC
CC
    peptides are not suitable as drug candidates as they require frequent
    administration due to rapid degradation by peptidases in the body.
CC
CC
    Modifying and attaching therapeutic peptides to albumin prevents or
CC
     reduces the action of peptidases to increase length of activity (half
    life) and specificity as bonding to large molecules decreases
CC
     intracellular uptake and interference with physiological processes.
CC
CC
    AAB90829 to AAB92441 represent peptides which can be used in the
CC
     exemplification of the present invention
XX
SO
     Sequence 24 AA;
                          85.4%; Score 35; DB 4; Length 24;
  Query Match
```

100.0%; Pred. No. 3.6;

Best Local Similarity

```
7; Conservative
                                                   0; Indels
                                                                 0; Gaps
                                                                              0:
 Matches
                                 0;
                                   Mismatches
Qу
            1 LVFFAED 7
              1 LVFFAED 7
Db
RESULT 69
AAB91805
ID
    AAB91805 standard; peptide; 24 AA.
XX
AC
    AAB91805;
XX
DT
    22-JUN-2001 (first entry)
XX
    Amyloid beta-protein fragment peptide SEQ ID NO:981.
DE
XX
    Protection; endogenous therapeutic peptide; peptidase; conjugation;
KW
KW
    blood component; modification; succinimidyl; maleimido group; amino;
    hydroxyl; thiol; hormone; growth factor; neurotransmitter.
KW
XX
OS
    Homo sapiens.
OS
    Synthetic.
XX
PN
    WO200069900-A2.
XX
PD
    23-NOV-2000.
XX
PF
    17-MAY-2000; 2000WO-US013576.
XX
PR
    17-MAY-1999;
                    99US-0134406P.
PR
    10-SEP-1999;
                    99US-0153406P.
    15-OCT-1999;
                    99US-0159783P.
PR
XX
     (CONJ-) CONJUCHEM INC.
PΑ
XX
     Bridon DP, Ezrin AM, Milner PG, Holmes DL, Thibaudeau K;
PΙ
XX
DR
    WPI; 2001-112059/12.
XX
    Modifying and attaching therapeutic peptides to albumin prevents
PT
PT
    peptidase degradation, useful for increasing length of in vivo activity.
XX
PS
    Disclosure; Page 515; 733pp; English.
XX
    The present invention describes a modified therapeutic peptide (I)
CC
CC
     comprising a therapeutically active amino acid region (III) and a
CC
     reactive group (II) (e.g. succinimidyl and maleimido groups) attached to
     a less therapeutically active amino acid region (IV), which covalently
CC
    bonds with amino/hydroxyl/thiol groups on blood components to form a
CC
    peptidase stabilised therapeutic peptide composed of 3-50 amino acids.
CC
CC
     (I) are useful for modifying therapeutic peptides e.g. hormones, growth
CC
     factors and neurotransmitters, to protect them from peptidase activity in
CC
     vivo for the treatment of various disorders. Endogenous therapeutic
CC
     peptides are not suitable as drug candidates as they require frequent
CC
     administration due to rapid degradation by peptidases in the body.
CC
    Modifying and attaching therapeutic peptides to albumin prevents or
```

```
reduces the action of peptidases to increase length of activity (half
CC
CC
     life) and specificity as bonding to large molecules decreases
CC
     intracellular uptake and interference with physiological processes.
CC
     AAB90829 to AAB92441 represent peptides which can be used in the
CC
     exemplification of the present invention
XX
SO
     Sequence 24 AA;
  Query Match
                          85.4%; Score 35; DB 4; Length 24;
  Best Local Similarity
                          100.0%; Pred. No. 3.6;
            7; Conservative
                               0; Mismatches
                                                   0; Indels
                                                                  0; Gaps
                                                                              0;
  Matches
            1 LVFFAED 7
Qy
              1111111
            1 LVFFAED 7
Db
RESULT 70
AAW47229
     AAW47229 standard; peptide; 26 AA.
XX
AC
    AAW47229;
XX
DT
     22-MAY-1998 (first entry)
XX
DE
     Beta-amyloid peptide residues 10-35.
XX
KW
     Screening assay; beta-amyloid peptide; treatment; amyloidosis disease;
KW
     Alzheimer's disease.
XX
OS
     Homo sapiens.
XX
PN
     US5721106-A.
XX
PD
     24-FEB-1998.
XX
                    94US-00304585.
ΡF
     12-SEP-1994;
XX
PR
     13-AUG-1991;
                    91US-00744767.
XX
PA
     (MINU ) UNIV MINNESOTA.
     (HARD ) HARVARD COLLEGE.
PΆ
XX
PΙ
     Mantyh PW, Maggio JE;
XX
     WPI; 1998-168404/15.
DR
XX
     New in vitro screening assay for Alzheimer's disease drugs - comprises
РΤ
     assessing binding of labelled beta-amyloid peptide to silk sample.
PT
XX
PS
     Claim 8; Col 31-32; 36pp; English.
XX
     The present sequence was used in the development of a novel in vitro
CC
     screening assay for agents capable of affecting the deposition of beta-
CC
CC
     amyloid peptide (BAP) on tissue. The method comprises contacting a silk
CC
     sample with labelled BAP, optionally in the presence of a test agent,
     detecting the amount of label bound to the silk and assessing the effect
CC
```

```
CC
     of the agent on the deposition of BAP. Agents that inhibit binding of BAP
CC
     to silk are potentially useful for treating amyloidosis diseases,
CC
     especially Alzheimer's disease
XX
SQ
     Sequence 26 AA;
  Query Match
                          85.4%; Score 35; DB 2; Length 26;
  Best Local Similarity
                          100.0%; Pred. No. 3.9;
            7; Conservative
                                 0; Mismatches
                                                                  0; Gaps
                                                                              0;
                                                   0; Indels
            1 LVFFAED 7
Qу
              Db
            8 LVFFAED 14
RESULT 71
AAY33408
    AAY33408 standard; peptide; 26 AA.
XX
AC
    AAY33408;
XX
DT
     03-DEC-1999 (first entry)
XX
DΕ
    Human amyloidogenic A-beta peptide 2.
XX
KW
     Amyloidogenic; beta-amyloid; A-beta peptide; human; inhibitor;
KW
     fibrillogenesis; amyloid plaque; amyloidosis; Alzheimer's disease;
KW
     Down's Syndrome.
XX
OS
    Homo sapiens.
XX
ΡN
    W09941279-A2.
XX
PD
    19-AUG-1999.
XX
PF
     12-FEB-1999;
                    99WO-US003231.
XX
PR
     13-FEB-1998;
                    98US-0074658P.
XX
PΑ
     (ARCH-) ARCH DEV CORP.
XX
PΙ
    Lynn DG, Meredith SC, Burkoth TS;
XX
DR
    WPI; 1999-561326/47.
XX
     Inhibiting amyloid plaque formation in humans suffering from amyloidosis,
PT
PT
    Alzheimer's disease or Down's Syndrome.
XX
PS
    Claim 22; Page 140; 141pp; English.
XX
CC
    This invention describes a novel method for inhibiting amyloid
CC
     fibrillogenesis which comprises contacting tissue with a composition
CC
     comprising an amyloidogenic peptide, beta-amyloid, that has been blocked
CC
     at an end terminal or a side chain, by conjugation to polyethylene
CC
     glycol, by conjugation to a second compound and a pharmaceutically
CC
     acceptable buffer, solvent or diluent. The methods are used to inhibit
CC
     amyloid plaque formation in humans suffering from amyloidosis,
```

```
CC
     Alzheimer's disease or Down's Syndrome. This sequence represents a
CC
     fragment of the beta-amyloid peptide described in the method of the
CC
     invention
XX
SQ
     Sequence 26 AA;
  Query Match
                          85.4%; Score 35; DB 2; Length 26;
  Best Local Similarity
                          100.0%; Pred. No. 3.9;
  Matches
            7; Conservative
                                 0; Mismatches
                                                    0; Indels
                                                                  0; Gaps
                                                                              0;
            1 LVFFAED 7
Qу
              1111111
Db
            8 LVFFAED 14
RESULT 72
AAB84431
     AAB84431 standard; peptide; 26 AA.
XX
AC
     AAB84431;
XX
DT
     22-AUG-2001 (first entry)
XX
DE
     Partial sequence of a human beta-amyloid precursor protein.
XX
KW
     Beta-amyloid precursor protein; APP; chimeric peptide; B cell epitope;
KW
     vaccine.
XX
OS
     Homo sapiens.
XX
ΡN
     W0200142306-A2.
XX
PD
     14-JUN-2001.
XX
PF
     08-DEC-2000; 2000WO-US033203.
XX
PR
     08-DEC-1999;
                    99US-0169687P.
XX
PΑ
     (MIND-) MINDSET BIOPHARMACEUTICALS USA INC.
XX
PΙ
     Chain B;
XX
     WPI; 2001-381648/40.
DR
XX
PT
     Novel chimeric peptide containing N- or C-terminal end-specific B cell
PT
     epitope from naturally occurring internal peptide cleavage product (such
PT
     as beta amyloid peptide) of a precursor protein, joined to T cell
PΤ
     epitope.
XX
PS
     Claim 3; Page 43; 47pp; English.
XX
CC
     The present sequence represents a partial sequence of a human beta-
CC
     amyloid precursor protein (APP). The peptide is used to create chimeric
CC
     peptides of the invention. The chimeric peptides contain a N- or C-
CC
     terminal end-specific B cell epitope from a naturally occurring internal
CC
     peptide cleavage product of a precursor or mature protein, as a free N-
CC
     or C-terminus, joined to a T cell epitope, with or without a spacer amino
```

```
CC
     acid residue. Chimeric peptides comprising betaAPP peptides slow down,
CC
     reduce or prevent the accumulation of amyloid beta peptide in the
CC
     extracellular space, interstitial fluid and cerebrospinal fluid of the
CC
     brain, and aggregation into senile amyloid deposits or plaques. They also
CC
     block the interaction of amyloid beta peptides with other molecules that
CC
     contribute the neurotoxicity of amyloid beta. The chimeric peptides are
CC
     useful for immunizing humans against the free N- or C-terminus of an
CC
     internal self peptide cleavage product (e.g. APP peptide) derived from a
     precursor protein or a mature protein. The internal peptide cleavage
CC
CC
     product is the self molecule of the mammal
XX
SQ
     Sequence 26 AA;
  Query Match
                          85.4%; Score 35; DB 4; Length 26;
  Best Local Similarity
                          100.0%; Pred. No. 3.9;
  Matches
            7; Conservative
                                0; Mismatches
                                                   0; Indels
                                                                 0;
                                                                     Gaps
                                                                              0;
            1 LVFFAED 7
QУ
              Db
            1 LVFFAED 7
RESULT 73
ABU63718
ID
     ABU63718 standard; peptide; 26 AA.
XX
AC
     ABU63718;
XX
DT
     15-OCT-2003 (first entry)
XX
DE
     Rat amyloid beta 1-40 (Abeta1-40) peptide insulysin cleavage product #11.
XX
KW
     Rat; amyloid beta; Abeta; amyloid fibril; amyloid plaque; neurotoxicity;
KW
     amyloid peptide-inactivating enzyme; hydrolysis; zinc metallopeptidase;
KW
     insulin degrading enzyme; IDE; insulysin; neprelysin; peptide therapy;
KW
     Alzheimer's disease; nootropic; neuroprotective.
XX
OS
     Rattus sp.
XX
PN
     US2003083277-A1.
XX
PD
     01-MAY-2003.
XX
PF
     26-FEB-2001; 2001US-00792079.
XX
PR
     24-FEB-2000; 2000US-0184826P.
XX
PΑ
     (HERS/) HERSH L B.
XX
PI
     Hersh LB;
XX
     WPI; 2003-576623/54.
DR
XX
PT
     Preventing formation or growth of amyloid fibrils or plaques without
PT
     causing neurotoxicity, useful for treating Alzheimer's disease, comprises
PT
     administering an amyloid peptide inactivating enzyme.
XX
```

```
Example 11; Page 9; 20pp; English.
XX
CC
     The invention discloses a method for preventing the formation or growth
CC
     of amyloid fibrils or plaques without causing neurotoxicity. The method
CC
     comprises administering an inactivation effective amount of an amyloid
CC
     peptide-inactivating enzyme to a mammal. The strategy is to hydrolyse the
     amyloid beta (Abeta) peptides before they form amyloid plaques using the
CC
     zinc metallopeptidase insulin degrading enzyme (IDE), insulysin or
CC
CC
     neprelysin. The methods and enzymes are useful for treating (e.g peptide
CC
     therapy) Alzheimer's disease. The enzymes are useful for inducing the
CC
     synthesis of endogenous amyloid inactivating enzymes, such as insulysin
CC
     or neprelysin, within the brain of the affected individuals. The sequence
CC
     presented is a Abeta1-40 peptide insulysin cleavage product
XX
SQ
     Sequence 26 AA;
  Query Match
                          85.4%; Score 35; DB 6; Length 26;
  Best Local Similarity
                          100.0%; Pred. No. 3.9;
  Matches
             7; Conservative
                               0; Mismatches
                                                   0; Indels
                                                                  0; Gaps
                                                                              0;
            1 LVFFAED 7
Qу
              111111
            3 LVFFAED 9
RESULT 74
     AAY33409 standard; peptide; 27 AA.
XX
AC
     AAY33409;
XX
DT
     03-DEC-1999 (first entry)
XX
DE
     Human amyloidogenic A-beta peptide C-terminal fragment.
XX
KW
     Amyloidogenic; beta-amyloid; A-beta peptide; human; inhibitor;
KW
     fibrillogenesis; amyloid plaque; amyloidosis; Alzheimer's disease;
KW
     Down's Syndrome.
XX
OS
     Homo sapiens.
XX
PN
     WO9941279-A2.
XX
PD
     19-AUG-1999.
XX
PF
     12-FEB-1999;
                    99WO-US003231.
XX
PR
     13-FEB-1998;
                    98US-0074658P.
XX
PΑ
     (ARCH-) ARCH DEV CORP.
XX
PΙ
     Lynn DG, Meredith SC,
                            Burkoth TS;
XX
DR
     WPI; 1999-561326/47.
XX
PT
     Inhibiting amyloid plaque formation in humans suffering from amyloidosis,
PT
     Alzheimer's disease or Down's Syndrome.
```

PS

```
XX
PS
     Disclosure; Page 141; 141pp; English.
XX
CC
     This invention describes a novel method for inhibiting amyloid
CC
     fibrillogenesis which comprises contacting tissue with a composition
CC
     comprising an amyloidogenic peptide, beta-amyloid, that has been blocked
CC
     at an end terminal or a side chain, by conjugation to polyethylene
CC
     glycol, by conjugation to a second compound and a pharmaceutically
CC
     acceptable buffer, solvent or diluent. The methods are used to inhibit
CC
     amyloid plaque formation in humans suffering from amyloidosis,
CC
     Alzheimer's disease or Down's Syndrome. This sequence represents the C-
CC
     terminal fragment of a PEG-derivatized beta-amyloid peptide described in
CC
     the method of the invention
XX
SQ
     Sequence 27 AA;
  Query Match
                          85.4%; Score 35; DB 2; Length 27;
  Best Local Similarity
                          100.0%; Pred. No. 4.1;
  Matches
            7; Conservative
                               0; Mismatches
                                                                 0; Gaps
                                                   0; Indels
                                                                              0;
            1 LVFFAED 7
Qy
              111111
            9 LVFFAED 15
RESULT 75
AAP70594
     AAP70594 standard; peptide; 28 AA.
XX
AC
     AAP70594;
XX
DT
     25-MAR-2003
                 (revised)
DT
     15-APR-1991
                  (first entry)
XX
DE
     Sequence of Alzheimer's amyloid polypeptide (AAP).
XX
KW
     Diagnosis; immunologic assay.
XX
OS
     Homo sapiens.
XX
ΡN
     US4666829-A.
XX
PD
     19-MAY-1987.
XX
PF
     15-MAY-1985;
                    85US-00734660.
XX
PR
     15-MAY-1985;
                    85US-00734660.
XX
PΑ
     (REGC ) UNIV CALIFORNIA.
XX
PΙ
     Glenner GG, Wong CW;
XX
DR
     WPI; 1987-157148/22.
XX
РΤ
     Alzheimer's amyloid polypeptide - used for obtaining antibodies and
PT
     nucleotide probes for diagnosis of Alzheimer's disease.
XX
```

```
PS
     Claim 1; Col 11; 8pp; English.
XX
CC
     Brains obtd. from patients suspected of having Alzheimer's disease and
CC
     exhibiting extensive cerebrovascular amyloidosis were used for AAP
CC
     isolation. The AAP can be used to obtain antibodies which can be used as
     reagents (claimed) in a blood or tissue immunologic assay for the
CC
CC
     disease. It can also be used to develop a probe (claimed) which can be
CC
     used in a diagnostic test (claimed). (Updated on 25-MAR-2003 to correct
     PA field.)
CC
XX
SQ
     Sequence 28 AA;
  Query Match
                          85.4%; Score 35; DB 1; Length 28;
  Best Local Similarity
                          100.0%; Pred. No. 4.2;
  Matches
             7; Conservative 0; Mismatches
                                                   0; Indels
                                                                  0;
                                                                     Gaps
                                                                              0;
            1 LVFFAED 7
Qy
              Db
           17 LVFFAED 23
RESULT 76
AAP90381
ID AAP90381 standard; protein; 28 AA.
XX
AC
     AAP90381;
XX
DΤ
     25-MAR-2003 (revised)
DT
     01-NOV-1989
                  (first entry)
XX
DΕ
     Synthetic A4 amyloid peptide.
XX
ΚW
     Synthetic; A4 amyloid polypeptide; Alzheimer's disease; immunoassays;
KW
     antibodies.
XX
OS
     Synthetic.
XX
PN
     WO8906242-A.
XX
     13-JUL-1989.
PD
XX
PF
     11-OCT-1988;
                    88WO-US003590.
XX
PR
     08-OCT-1987;
                    87US-00105751.
XX
PΑ
     (MCLE-) MCLEAN HOSPITAL CORP.
PA
     (UYRP ) UNIV ROCHESTER.
XX
PI
     Majocha R, Marotta CA,
                             Zain S;
XX
DR
     WPI; 1989-220551/30.
XX
PT
     Antibodies to A4 amyloid polypeptide - used in immunoassays and for
     imaging of A4-amyloid in Alzheimer's diseased patients.
PT
XX
PS
     Claim 1; Page 27; 30pp; English.
XX
```

```
CC
     Synthetic A4 amyloid polypeptide (see also AAP90382, AAP90383). Used as
CC
     immunogen, (un)coupled, or to produce antibodies. Used in immunoassays
CC
     and for imaging of A4 amyloid in Alzheimer's disease. (Updated on 25-MAR-
CC
     2003 to correct PA field.)
XX
SQ
     Sequence 28 AA;
  Query Match
                          85.4%; Score 35; DB 1; Length 28;
  Best Local Similarity 100.0%; Pred. No. 4.2;
            7; Conservative 0; Mismatches
                                                   0; Indels
                                                                  0; Gaps
                                                                              0;
            1 LVFFAED 7
QУ
              111111
           17 LVFFAED 23
Db
RESULT 77
AAR60368
    AAR60368 standard; peptide; 28 AA.
ID
XX
AC
    AAR60368;
XX
DT
    25-MAR-2003 (revised)
DT
    15-MAR-1995 (first entry)
XX
DE
    Beta-amyloid (1-28).
XX
KW
    Amyloid precursor protein; APP; Alzheimer's disease; beta-amyloid;
KW
     anti-beta-amyloid antibody; diagnosis; immunogen; antigen; epitope.
XX
OS
    Homo sapiens.
XX
PN
    WO9417197-A1.
XX
PD
     04-AUG-1994.
XX
PF
     24-JAN-1994;
                    94WO-JP000089.
XX
PR
     25-JAN-1993;
                    93JP-00010132.
PR
     05-FEB-1993;
                    93JP-00019035.
PR
     16-NOV-1993;
                    93JP-00286985.
    28-DEC-1993;
                    93JP-00334773.
PR
XX
PΑ
     (TAKE ) TAKEDA CHEM IND LTD.
XX
PΙ
     Suzuki N, Odaka A, Kitada C;
XX
DR
    WPI; 1994-264110/32.
XX
     Antibodies recognising specific parts of beta-amyloid - can be used for
PT
PT
     diagnosis of diseases implicating beta-amyloid, such as Alzheimer's
PT
     disease.
XX
PS
     Claim 7; Page 84; 116pp; Japanese.
XX
CC
    Antibodies which recognise specific subfragments of the beta-amyloid
CC
     protein are claimed. Specifically, the antibodies (which are pref.
```

```
CC
     monoclonal) recognise residues 1-16 and/or 1-28 from the N-terminal
CC
     portion of beta-amyloid or they recognise residues 25-35 or 35-43 from
CC
     the C-terminal portion. The antibodies are useful for assaying beta-
CC
     amyloid and its derivatives for diagnosis of Alzheimer's disease.
CC
     (Updated on 25-MAR-2003 to correct PN field.)
XX
SO
     Sequence 28 AA;
  Query Match
                          85.4%; Score 35; DB 2; Length 28;
  Best Local Similarity
                          100.0%; Pred. No. 4.2;
             7; Conservative
                               0; Mismatches
                                                    0; Indels
                                                                  0; Gaps
                                                                              0;
            1 LVFFAED 7
Qy
              Db
           17 LVFFAED 23
RESULT 78
AAR54702
     AAR54702 standard; peptide; 28 AA.
XX
AC
     AAR54702;
XX
DT
     25-MAR-2003 (revised)
     15-DEC-1994 (first entry)
DT
XX
DΕ
     Beta-amyloid fragment (1-28).
XX
KW
     Beta-amyloid protein; BAP; Alzheimer's disease; diagnosis.
XX
os
     Homo sapiens.
XX
ΡN
     WO9409364-A1.
XX
PD
     28-APR-1994.
XX
PF
     13-OCT-1993;
                    93WO-US009772.
XX
PR
     13-OCT-1992;
                    92US-00959251.
XX
PΑ
     (UYDU-) UNIV DUKE.
XX
PI
     Strittmatter WJ;
XX
DR
     WPI; 1994-151484/18.
XX
PT
     Immobilised beta-amyloid protein or fragments - used in assays for
     obtaining prods for use in the diagnosis and treatment of disorders such
PT
PΤ
     as Alzheimer's disease.
XX
_{\mathrm{PS}}
     Claim 4; Page 28; 49pp; English.
XX
CC
     A construct comprising a beta-amyloid protein (BAP) or fragment (esp. the
CC
     peptides given in AAR54702-03) immobilised on a solid support can be used
     to detect cpds. which bind to BAP. Binding of proteins in human
CC
CC
     cerebrospinal fluid proteins were shown to bind to beta- amyloid peptides
     1-28 and 12-28. Hydropathic mimic peptide (12-28) was used as control.
CC
```

```
CC
     (Updated on 25-MAR-2003 to correct PN field.)
XX
SQ
     Sequence 28 AA;
  Query Match
                          85.4%; Score 35; DB 2;
                                                    Length 28;
  Best Local Similarity
                          100.0%; Pred. No. 4.2;
             7; Conservative
                                 0; Mismatches
                                                    0; Indels
                                                                              0;
                                                                  0; Gaps
            1 LVFFAED 7
Qу
              Db
           17 LVFFAED 23
RESULT 79
AAR64171
     AAR64171 standard; peptide; 28 AA.
ID
XX
AC
     AAR64171;
XX
DT
     25-MAR-2003
                  (revised)
DT
     03-AUG-1995
                  (first entry)
XX
DE
     A4-P(1-28) a partial beta amyloid peptide.
XX
KW
     beta amyloid protein; mutant; variant; detection; amyloid deposition;
KW
     diagnosis; amyloidosis associated disease; Alzheimer's disease;
     Down's syndrome; A4-P(1-28).
KW
XX
OS
     Synthetic.
XX
PN
     WO9428412-A1.
XX
PD
     08-DEC-1994.
XX
PF
     27-MAY-1994;
                    94WO-US005809.
XX
PR
     28-MAY-1993;
                    93US-00069010.
XX
PΑ
     (MIRI-) MIRIAM HOSPITAL.
XX
PΙ
     Marotta CA, Majocha RE;
XX
DR
     WPI; 1995-023013/03.
XX
PT
     Amyloid binding composition comprising labelled amyloid protein and
PT
     carrier - useful for in vivo imaging of amyloid deposits, for diagnosing
PT
     Alzheimer's disease and Down's Syndrome.
XX
PS
     Example 3; Page 23; 58pp; English.
XX
CC
     AAR64171, the A4-P(1-28) polypeptide is deriv. from vascular amyloid of
CC
     the AD (Alzheimer's disease) brain and a Down Syndrome brain. Three of
CC
     the 28 amino acids are different from the A4-O(1-28) peptide shown in
CC
     AAR64170. A4-O has strong aggregation properties, and binds to itself
CC
     strongly. It is used to obtain and select beta amyloid proteins that can
CC
     be used for in vivo imaging of amyloid deposits and hence diagnosis of an
CC
     amyloidosis-associated disease, such as AD or Down's syndrome. AAR64165
```

```
shows the generic sequence of the amyloid protein for generation of
CC
CC
     variants. (Updated on 25-MAR-2003 to correct PN field.)
XX
SQ
     Sequence 28 AA;
                          85.4%; Score 35; DB 2; Length 28; 100.0%; Pred. No. 4.2;
  Query Match
  Best Local Similarity
                               0; Mismatches
 Matches
             7; Conservative
                                                     0; Indels
                                                                                0;
                                                                   0; Gaps
            1 LVFFAED 7
Qу
              111111
Db
           17 LVFFAED 23
RESULT 80
AAR64164
    AAR64164 standard; peptide; 28 AA.
XX
AC
     AAR64164;
XX
DT
     25-MAR-2003
                   (revised)
DT
     02-AUG-1995
                  (first entry)
XX
DE
     Generic beta amyloid protein variant.
XX
KW
     generic sequence; beta amyloid protein; mutant; variant; detection;
KW
     amyloid deposition; diagnosis; amyloidosis associated disease;
     Alzheimer's disease; Down's syndrome.
KW
XX
OS
     Synthetic.
XX
FH
                     Location/Qualifiers
     Key
FT
     Misc-difference 11
FT
                     /note= "Glu or Gln"
FT
     Misc-difference 27
FT
                     /note= "Ser or Asn"
FT
     Misc-difference 28
FT
                     /note= "Ala or Lys"
XX
PN
     WO9428412-A1.
XX
     08-DEC-1994.
PD
XX
     27-MAY-1994;
                    94WO-US005809.
PF
XX
PR
     28-MAY-1993;
                    93US-00069010.
XX
     (MIRI-) MIRIAM HOSPITAL.
PA
XX
PΙ
     Marotta CA, Majocha RE;
XX
     WPI; 1995-023013/03.
DR
XX
     Amyloid binding composition comprising labelled amyloid protein and
PT
PT
     carrier - useful for in vivo imaging of amyloid deposits, for diagnosing
PT
     Alzheimer's disease and Down's Syndrome.
XX
```

```
Claim 3; Page 42; 58pp; English.
PS
XX
CC
     AAR64164 shows the generic amino acid sequence of a variant beta amyloid
CC
    protein. The protein binds amyloid and is useful for in vivo imaging of
CC
     amyloid deposits and hence diagnosis of an amyloidosis-associated
     disease, such as Alzheimer's disease or Down's syndrome. AAR64165-69 show
CC
CC
     specifc variants generated from this generic sequence with addition amino
CC
     acids. (Updated on 25-MAR-2003 to correct PN field.)
XX
SQ
     Sequence 28 AA;
                          85.4%; Score 35; DB 2; Length 28;
  Query Match
                          100.0%; Pred. No. 4.2;
 Best Local Similarity
            7; Conservative
                               0; Mismatches
 Matches
                                                   0; Indels
                                                                 0; Gaps
                                                                             0;
            1 LVFFAED 7
Qу
              Db
           17 LVFFAED 23
RESULT 81
AAR64172
ID
    AAR64172 standard; peptide; 28 AA.
XX
AC
    AAR64172;
XX
DT
    25-MAR-2003
                  (revised)
DT
    03-AUG-1995
                 (first entry)
XX
DE
    A4-B(1-28) a partial beta amyloid peptide.
XX
KW
    beta amyloid protein; mutant; variant; detection; amyloid deposition;
KW
    diagnosis; amyloidosis associated disease; Alzheimer's disease;
     Down's syndrome; A4-B(1-28).
KW
XX
OS
     Synthetic.
XX
PN
    WO9428412-A1.
XX
    08-DEC-1994.
PD
XX
ΡF
    27-MAY-1994;
                   94WO-US005809.
XX
PR
    28-MAY-1993;
                   93US-00069010.
XX
     (MIRI-) MIRIAM HOSPITAL.
PA
XX
PΙ
    Marotta CA, Majocha RE;
XX
    WPI; 1995-023013/03.
DR
XX
PT
    Amyloid binding composition comprising labelled amyloid protein and
PT
     carrier - useful for in vivo imaging of amyloid deposits, for diagnosing
PT
     Alzheimer's disease and Down's Syndrome.
XX
PS
     Example 3; Page 23; 58pp; English.
XX
```

```
CC
    AAR64172, the A4-B(1-28) polypeptide is deriv. from vascular amyloid of
CC
     the AD (Alzheimer's disease) brain and a Down Syndrome brain. Three of
CC
     the 28 amino acids are different from the A4-O(1-28) peptide shown in
CC
    AAR64170. A4-0 has strong aggregation properties, and binds to itself
CC
     strongly. It is used to obtain and select beta amyloid proteins that can
CC
     be used for in vivo imaging of amyloid deposits and hence diagnosis of an
CC
     amyloidosis-associated disease, such as AD or Down's syndrome. AAR64165
CC
     shows the generic sequence of the amyloid protein for generation of
CC
     variants. (Updated on 25-MAR-2003 to correct PN field.)
XX
SQ
     Sequence 28 AA;
  Query Match
                          85.4%; Score 35; DB 2; Length 28;
                                   Pred. No. 4.2;
  Best Local Similarity
                          100.0%;
 Matches
             7; Conservative 0; Mismatches
                                                   0; Indels
                                                                  0; Gaps
                                                                              0;
            1 LVFFAED 7
Qу
              111111
Db
           17 LVFFAED 23
RESULT 82
AAR64170
     AAR64170 standard; peptide; 28 AA.
XX
AC
    AAR64170;
XX
DT
    25-MAR-2003 (revised)
DT
    03-AUG-1995
                 (first entry)
XX
DE
    A4-O(1-28) a partial beta amyloid peptide.
XX
KW
    beta amyloid protein; mutant; variant; detection; amyloid deposition;
KW
     diagnosis; amyloidosis associated disease; Alzheimer's disease;
KW
     Down's syndrome; A4-O(1-28).
XX
OS
     Synthetic.
XX
PN
    WO9428412-A1.
XX
PD
    08-DEC-1994.
XX
PF
     27-MAY-1994;
                    94WO-US005809.
XX
PR
    28-MAY-1993;
                    93US-00069010.
XX
PA
     (MIRI-) MIRIAM HOSPITAL.
XX
PI
    Marotta CA, Majocha RE;
XX
DR
    WPI; 1995-023013/03.
XX
PT
    Amyloid binding composition comprising labelled amyloid protein and
PT
     carrier - useful for in vivo imaging of amyloid deposits, for diagnosing
PT
     Alzheimer's disease and Down's Syndrome.
XX
PS
     Example 1; Page 23; 58pp; English.
```

```
XX
CC
    AAR64170, the A4-O(1-28) polypeptide is the first 28 amino acids of the
CC
     4.2 kD peptide deriv. from senile plaque cores of an AD (Alzheimer's
CC
    disease) brain, known as beta amyloid. A4-0 has strong aggregation
CC
    properties, and binds to itself strongly. This peptide is used to obtain
CC
     and select beta amyloid proteins that can be used for in vivo imaging of
CC
     amyloid deposits and hence diagnosis of an amyloidosis-associated
    disease, such as AD or Down's syndrome. AAR64165 shows the generic
CC
CC
     sequence of the amyloid protein for generation of variants. (Updated on
CC
     25-MAR-2003 to correct PN field.)
XX
SQ
    Sequence 28 AA;
  Query Match
                          85.4%; Score 35; DB 2; Length 28;
 Best Local Similarity
                         100.0%; Pred. No. 4.2;
 Matches
            7; Conservative
                               0; Mismatches
                                                   0; Indels
                                                                 0; Gaps
                                                                             0;
            1 LVFFAED 7
Qу
              Db
           17 LVFFAED 23
RESULT 83
AAW01413
TD
    AAW01413 standard; protein; 28 AA.
XX
AC
    AAW01413;
XX
DT
    20-JAN-1997 (first entry)
XX
DE
    Beta/A4-amyloid peptide residues 1-28.
XX
KW
     Beta/A4-amyloid peptide; tissue plasminogen activator;
KW
    Alzheimer's disease; stimulation; investigation; pathogenesis;
KW
    hereditary cerebral haemorrhage with amyloidosis-Dutch type; control;
KW
     cerebral amyloid angiopathy; cerebral; haemorrhage; hemorrhage.
XX
OS
    Homo sapiens.
XX
PN
    WO9615799-A1.
XX
PD
     30-MAY-1996.
XX
PF
                    95WO-US015007.
    22-NOV-1995;
XX
PR
                   94US-00347144.
    22-NOV-1994;
XX
PΑ
     (RUTF ) UNIV RUTGERS STATE NEW JERSEY.
XX
PΙ
    Anderson S;
XX
    WPI; 1996-268332/27.
DR
XX
PT
     Use of agents which bind beta-amyloid peptide - for diagnosis, prevention
PT
     and treatment of vascular damage caused by amyloid deposits, partic. in
PT
     haemorrhaging and Alzheimer's disease.
XX
```

```
PS
    Example 1; Fig 1; 52pp; English.
XX
CC
    To investigate the effects of beta-amyloid peptide (BAP) on tissue
CC
    plasminogen activator (t-PA) 3 synthetic peptides were used. One peptide
CC
     contained 42 amino acids and corresp. to the full length BAP (AAR95248).
    The other 2 peptides (AAR95249 and 50) contained the 28 N-terminal
CC
CC
     residues of the BAP found in Alzheimer's disease and hereditary cerebral
    haemorrhage with amyloidosis-Dutch type (HCHWA-D), respectively. In an
CC
CC
    assay to determine the effect of the peptides on t-PA activation, each
CC
    peptide (AAR95248, 49 and 50) gave 1st order rate constant of activation
CC
     (k(app)) values of 13.4, 13.9 and 14.5, respectively, compared to 1.7 and
CC
     7.8 for nill and fibrinogen controls. The results demonstrate that the
    \ensuremath{\mathtt{BAP}} are able to stimulate t-PA activity in vitro, which is significant in
CC
CC
    that it provides a means for investigating and controlling the
CC
    pathogenesis of Alzheimer's disease, HCHWA-D and cerebral amyloid
CC
     angiopathy related cerebral haemorrhage
XX
SQ
     Sequence 28 AA;
  Query Match
                          85.4%; Score 35; DB 2; Length 28;
                          100.0%; Pred. No. 4.2;
 Best Local Similarity
 Matches
            7; Conservative
                                 0; Mismatches
                                                    0; Indels
                                                                  0; Gaps
                                                                              0;
Qу
            1 LVFFAED 7
              Db
           17 LVFFAED 23
RESULT 84
AAY39805
    AAY39805 standard; peptide; 28 AA.
XX
AC
    AAY39805;
XX
DT
     29-NOV-1999
                  (first entry)
XX
DΕ
     Beta-amyloid protein, Beta/A4 amyloid (1-28).
XX
KW
     Beta-amyloid protein; Alzheimer's disease; amyloidosis; joint swelling;
KW
     long-standing inflammation; malignancy; Familial Mediterranean Fever;
    multiple myeloma; plasma cell dyscrasia; long-term haemodialysis; kuru;
KW
     carpal tunnel syndrome; multiple spontaneous fracture; radiolucency;
KW
KW
     endocrine tumour; medullary carcinoma; Down's syndrome; scrapie;
KW
     Creutzfeldt-Jakob disease; Gerstmann Strausiler Syndrome;
KW
     subacute spongiform encephalopathy; therapy.
XX
OS
    Homo sapiens.
XX
PN
    US5958883-A.
XX
PD
     28-SEP-1999.
XX
     05-JUN-1995;
PF
                    95US-00461216.
XX
PR
     23-SEP-1992;
                    92US-00950417.
PR
     23-OCT-1992;
                    92US-00969734.
XX
```

```
PΑ
     (UNIW ) UNIV WASHINGTON.
XX
PΙ
     Snow AD;
XX
DR
     WPI; 1999-561062/47.
XX
PT
     Peptides of 6-8 amino acids useful for treating or preventing
PT
     amyloidosis.
XX
PS
     Disclosure; Col 67-68; 83pp; English.
XX
     This sequence represents a fragment of the beta-amyloid protein. The
CC
     invention relates to a method for treating or preventing a form of
CC
     amyloidosis, including Alzheimer's disease using this sequence. The
CC
CC
     compositions may be useful for treating or preventing the amyloidosis
     associated with long-standing inflammation, various forms of malignancy
CC
     (including B-cell type malignancies), Familial Mediterranean Fever,
CC
     multiple myeloma, plasma cell dyscrasias, long-term haemodialysis, carpal
CC
CC
     tunnel syndrome, joint swelling, multiple spontaneous fractures,
     radiolucency in the wrist and hip, endocrine tumours, medullary carcinoma
CC
     of the thyroid, diabetes, Alzheimer's disease, Down's syndrome,
CC
     Creutzfeldt-Jakob disease, Gerstmann Strausiler Syndrome, kuru, scrapie
CC
     and other subacute spongiform encephalopathies
CC
XX
SQ
     Sequence 28 AA;
                          85.4%; Score 35; DB 2; Length 28;
  Query Match
                          100.0%; Pred. No. 4.2;
  Best Local Similarity
             7; Conservative 0; Mismatches
                                                   0; Indels
                                                                  0; Gaps
                                                                              0;
  Matches
            1 LVFFAED 7
Qу
              Db
           17 LVFFAED 23
RESULT 85
AAW81467
     AAW81467 standard; peptide; 28 AA.
ID
XX
AC
     AAW81467;
XX
DT
     28-JAN-1999 (first entry)
XX
     Synthetic amyloid beta (Abeta) peptide 2 (residues 1-28).
DE
XX
ΚW
     Amyloid beta; Abeta; deoxygenated solvent; evaporative deposition;
     research; neurotoxicity; free-radical; glutamine synthetase.
KW
XX
OS
     Synthetic.
XX
PN
     US5840838-A.
XX
PD
     24-NOV-1998.
XX
PF
     29-FEB-1996;
                    96US-00609090.
XX
PR
     29-FEB-1996;
                    96US-00609090.
```

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XX
     (KENT ) UNIV KENTUCKY RES FOUND.
PA
XX
     Aksenov M, Carney JM, Hensley K, Butterfield DA;
PI
XX.
DR
     WPI; 1999-034120/03.
XX
     Process for treating synthetic amyloid beta peptides - by organic solvent
PT
     treatment, useful for studying neurotoxicity.
PT
XX
PS
     Claim 5; Col 9-10; 14pp; English.
XX
     Sequences AAW81466 to AAW81476 represent synthetic amyloid beta (Abeta)
CC
     peptides. The invention provides a process for treating a synthetic Abeta
CC
     peptide that comprises dissolving the peptide in a deoxygenated solvent
CC
     selected from trifluoroethanol, hexafluorocyclohexane, dimethyl
CC
     sulphoxide, morpholinopropanesulphonic acid, dimethylformamide and
CC
     acetonitrile to a concentration of 0.01-10 mg/ml, incubating the solution
CC
     at 20-65 deg. C for 0.5-4 hour, and removing the solvent by ''evaporative
CC
     deposition'' in 5-10 minutes. Synthetic amyloid beta peptides are useful
CC
     as research tools for studying neurotoxicity resulting from Abeta peptide
CC
     -enhanced free-radical production. The treatment increases the activity
CC
     of the synthetic Abeta peptides in tests to determine free-radical
CC
     generating capacity and glutamine synthetase inactivation
CC
XX
SO
     Sequence 28 AA;
                          85.4%; Score 35; DB 2;
                                                   Length 28;
  Query Match
  Best Local Similarity
                          100.0%; Pred. No. 4.2;
                                                                              0:
             7; Conservative 0; Mismatches
                                                   0;
                                                       Indels
                                                                  0; Gaps
  Matches
            1 LVFFAED 7
Qу
              111111
Db
           17 LVFFAED 23
RESULT 86
AAB35591
     AAB35591 standard; peptide; 28 AA.
XX
AC
     AAB35591;
XX
DT
     15-FEB-2001 (first entry)
XX
     Human clone D1N B(1-28) amyloid B peptide.
DE
XX
     Beta-amyloid; amyloid deposit; Alzheimer's disease; thrombolytic therapy;
KW
KW
     acute cardiovascular disease; therapy.
XX
OS
     Homo sapiens.
XX
     US6136548-A.
PN
XX
PD
     24-OCT-2000.
XX
PF
     02-SEP-1999;
                    99US-00388890.
XX
```

```
PR
     22-NOV-1994;
                    94US-00347144.
PR
     22-NOV-1995;
                    95WO-US015007.
PR
     26-JUL-1996;
                    96US-00686959.
XX
PΑ
     (RUTF ) UNIV RUTGERS STATE NEW JERSEY.
XX
PΙ
     Anderson S;
XX
DR
     WPI; 2001-030939/04.
XX
     Identifying mutant tissue-type plasminogen activator (t-PA) for improving
PT
     thrombolytic therapy or treating vascular hemorrhaging, by determining
PΤ
     whether t-PA binds to fibrin but not to a beta amyloid peptide.
PT
XX
PS
     Example 3; Col 26; 23pp; English.
XX
     The present invention describes a method for identifying mutant
CC
     derivatives of tissue-type plasminogen activator, which involves
CC
     determining whether or not they bind to beta-amyloid peptides and fibrin.
CC
     Mutants will only bind to the latter. These mutants are useful in
CC
     improved thrombolytic therapies, in the treatment of Alzheimer's disease
CC
     and in the treatment of acute cardiovascular disease, which may be caused
CC
     by myocardial infarction, stroke, ischaemia and pulmonary embolism
CC
XX
SQ
     Sequence 28 AA;
                          85.4%; Score 35; DB 4; Length 28;
  Query Match
                          100.0%; Pred. No. 4.2;
  Best Local Similarity
            7; Conservative 0; Mismatches
                                                                              0;
                                                   0; Indels
                                                                  0; Gaps
  Matches
            1 LVFFAED 7
Qу
              +1111111
           17 LVFFAED 23
Dh
RESULT 87
AAB35595
     AAB35595 standard; peptide; 28 AA.
ID
XX
     AAB35595;
AC
XX
DT
     15-FEB-2001 (first entry)
XX
     Human clone D7Q B(1-28) amyloid B peptide.
DE
XX
     Beta-amyloid; amyloid deposit; Alzheimer's disease; thrombolytic therapy;
KW
     acute cardiovascular disease; therapy.
KW
XX
OS
     Homo sapiens.
XX
PN
     US6136548-A.
XX
PD
     24-OCT-2000.
XX
ΡF
     02-SEP-1999;
                    99US-00388890.
XX
                    94US-00347144.
PR
     22-NOV-1994;
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95WO-US015007.
PR
     22-NOV-1995;
     26-JUL-1996;
                    96US-00686959.
PR
XX
     (RUTF ) UNIV RUTGERS STATE NEW JERSEY.
PΑ
XX
PΙ
     Anderson S;
XX
     WPI; 2001-030939/04.
DR
XX
     Identifying mutant tissue-type plasminogen activator (t-PA) for improving
PΤ
     thrombolytic therapy or treating vascular hemorrhaging, by determining
PT
     whether t-PA binds to fibrin but not to a beta amyloid peptide.
PT
XX
     Example 3; Col 26; 23pp; English.
PS
XX
     The present invention describes a method for identifying mutant
CC
     derivatives of tissue-type plasminogen activator, which involves
CC
     determining whether or not they bind to beta-amyloid peptides and fibrin.
CC
     Mutants will only bind to the latter. These mutants are useful in
CC
     improved thrombolytic therapies, in the treatment of Alzheimer's disease
CC
     and in the treatment of acute cardiovascular disease, which may be caused
CC
     by myocardial infarction, stroke, ischaemia and pulmonary embolism
CC
XX
     Sequence 28 AA;
SQ
                          85.4%; Score 35; DB 4; Length 28;
  Query Match
                          100.0%; Pred. No. 4.2;
  Best Local Similarity
            7; Conservative 0; Mismatches
                                                                      Gaps
                                                                              0;
  Matches
                                                    0; Indels
            1 LVFFAED 7
Qу
              1111111
           17 LVFFAED 23
Db
RESULT 88
AAB35594
     AAB35594 standard; peptide; 28 AA.
XX
AC
     AAB35594;
XX
     15-FEB-2001 (first entry)
DT
XX
     Human clone H6Q B(1-28) amyloid B peptide.
DE
XX
     Beta-amyloid; amyloid deposit; Alzheimer's disease; thrombolytic therapy;
KW
     acute cardiovascular disease; therapy.
KW
XX
OS
     Homo sapiens.
XX
PN
     US6136548-A.
XX
     24-OCT-2000.
PD
XX
     02-SEP-1999;
                     99US-00388890.
PF
XX
                     94US-00347144.
PR
     22-NOV-1994;
                    95WO-US015007.
PR
     22-NOV-1995;
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PR
     26-JUL-1996;
                    96US-00686959.
XX
PΑ
     (RUTF ) UNIV RUTGERS STATE NEW JERSEY.
XX
PΙ
    Anderson S;
XX
     WPI; 2001-030939/04.
DR
XX
     Identifying mutant tissue-type plasminogen activator (t-PA) for improving
PT
     thrombolytic therapy or treating vascular hemorrhaging, by determining
PT
     whether t-PA binds to fibrin but not to a beta amyloid peptide.
PT
XX
PS
     Example 3; Col 26; 23pp; English.
XX
     The present invention describes a method for identifying mutant
CC
     derivatives of tissue-type plasminogen activator, which involves
CC
     determining whether or not they bind to beta-amyloid peptides and fibrin.
CC
     Mutants will only bind to the latter. These mutants are useful in
CC
     improved thrombolytic therapies, in the treatment of Alzheimer's disease
CC
     and in the treatment of acute cardiovascular disease, which may be caused
CC
     by myocardial infarction, stroke, ischaemia and pulmonary embolism
CC
XX
SQ
     Sequence 28 AA;
                          85.4%; Score 35; DB 4; Length 28;
  Query Match
  Best Local Similarity
                          100.0%; Pred. No. 4.2;
                                                                              0;
                               0; Mismatches
                                                                  0; Gaps
  Matches
            7; Conservative
                                                    0; Indels
            1 LVFFAED 7
Qу
              111111
           17 LVFFAED 23
Db
RESULT 89
AAB35592
     AAB35592 standard; peptide; 28 AA.
ID
XX
AC
     AAB35592;
XX
     15-FEB-2001 (first entry)
DT
XX
     Human clone E3Q B(1-28) amyloid B peptide.
DE
XX
     Beta-amyloid; amyloid deposit; Alzheimer's disease; thrombolytic therapy;
KW
     acute cardiovascular disease; therapy.
ΚW
XX
OS
     Homo sapiens.
XX
PN
     US6136548-A.
XX
PD
     24-OCT-2000.
XX
                    99US-00388890.
PF
     02-SEP-1999;
XX
                    94US-00347144.
     22-NOV-1994;
PR
                    95WO-US015007.
     22-NOV-1995;
PR
                    96US-00686959.
PR
     26-JUL-1996;
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XX
     (RUTF ) UNIV RUTGERS STATE NEW JERSEY.
PΑ
XX
PI
     Anderson S;
XX
DR
     WPI; 2001-030939/04.
XX
     Identifying mutant tissue-type plasminogen activator (t-PA) for improving
PT
     thrombolytic therapy or treating vascular hemorrhaging, by determining
PT
     whether t-PA binds to fibrin but not to a beta amyloid peptide.
РΤ
XX
     Example 3; Col 26; 23pp; English.
PS
XX
     The present invention describes a method for identifying mutant
CC
     derivatives of tissue-type plasminogen activator, which involves
CC
     determining whether or not they bind to beta-amyloid peptides and fibrin.
CC
     Mutants will only bind to the latter. These mutants are useful in
CC
     improved thrombolytic therapies, in the treatment of Alzheimer's disease
CC
     and in the treatment of acute cardiovascular disease, which may be caused
CC
     by myocardial infarction, stroke, ischaemia and pulmonary embolism
CC
XX
     Sequence 28 AA;
SQ
                          85.4%; Score 35; DB 4; Length 28;
  Query Match
  Best Local Similarity
                          100.0%; Pred. No. 4.2;
                                                                              0;
                                                                  0; Gaps
             7; Conservative
                                 0; Mismatches
                                                    0; Indels
  Matches
            1 LVFFAED 7
Qу
              17 LVFFAED 23
Db
RESULT 90
AAB35593
     AAB35593 standard; peptide; 28 AA.
ID
XX
AC
     AAB35593;
XX
DT
     15-FEB-2001 (first entry)
XX
     Human clone R5Q B(1-28) amyloid B peptide.
DE
XX
     Beta-amyloid; amyloid deposit; Alzheimer's disease; thrombolytic therapy;
KW
     acute cardiovascular disease; therapy.
KW
XX
OS
     Homo sapiens.
XX
PN
     US6136548-A.
XX
PD
     24-OCT-2000.
XX
                     99US-00388890.
PF.
     02-SEP-1999;
XX
                     94US-00347144.
PR
     22-NOV-1994;
                     95WO-US015007.
PR
     22-NOV-1995;
                     96US-00686959.
PR
     26-JUL-1996;
XX
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(RUTF ) UNIV RUTGERS STATE NEW JERSEY.
PΑ
XX
     Anderson S;
PΙ
XX
     WPI; 2001-030939/04.
DR
XX
     Identifying mutant tissue-type plasminogen activator (t-PA) for improving
PT
     thrombolytic therapy or treating vascular hemorrhaging, by determining
PT
     whether t-PA binds to fibrin but not to a beta amyloid peptide.
PT
XX
     Example 3; Col 26; 23pp; English.
PS
XX
     The present invention describes a method for identifying mutant
CC
     derivatives of tissue-type plasminogen activator, which involves
CC
     determining whether or not they bind to beta-amyloid peptides and fibrin.
CC
     Mutants will only bind to the latter. These mutants are useful in
CC
     improved thrombolytic therapies, in the treatment of Alzheimer's disease
CC
     and in the treatment of acute cardiovascular disease, which may be caused
CC
     by myocardial infarction, stroke, ischaemia and pulmonary embolism
CC
XX
SQ
     Sequence 28 AA;
                          85.4%; Score 35; DB 4; Length 28;
  Query Match
                          100.0%; Pred. No. 4.2;
  Best Local Similarity
                                                                              0;
            7; Conservative 0; Mismatches / 0; Indels
                                                                  0; Gaps
  Matches
            1 LVFFAED 7
Qу
              111111
           17 LVFFAED 23
Db
RESULT 91
AAB35597
     AAB35597 standard; peptide; 28 AA.
ID
XX
     AAB35597;
AC
XX
DT
     15-FEB-2001 (first entry)
XX
     Human clone H13Q B(1-28) amyloid B peptide.
DE
XX
     Beta-amyloid; amyloid deposit; Alzheimer's disease; thrombolytic therapy;
KW
     acute cardiovascular disease; therapy.
KW
XX
     Homo sapiens.
OS
XX
PN
     US6136548-A.
XX
PD
     24-OCT-2000.
XX
PF
     02-SEP-1999;
                    99US-00388890.
XX
                    94US-00347144.
PR
     22-NOV-1994;
                     95WO-US015007.
 PR
     22-NOV-1995;
                    96US-00686959.
     26-JUL-1996;
 PR
 XX
      (RUTF ) UNIV RUTGERS STATE NEW JERSEY.
 PΑ
```

```
XX
PΙ
     Anderson S;
XX
DR
     WPI; 2001-030939/04.
XX
     Identifying mutant tissue-type plasminogen activator (t-PA) for improving
PT
PΤ
     thrombolytic therapy or treating vascular hemorrhaging, by determining
     whether t-PA binds to fibrin but not to a beta amyloid peptide.
PT
XX
PS
     Example 3; Col 26; 23pp; English.
XX
CC
     The present invention describes a method for identifying mutant
     derivatives of tissue-type plasminogen activator, which involves
CC
CC
     determining whether or not they bind to beta-amyloid peptides and fibrin.
     Mutants will only bind to the latter. These mutants are useful in
CC
     improved thrombolytic therapies, in the treatment of Alzheimer's disease
CC
     and in the treatment of acute cardiovascular disease, which may be caused
CC
     by myocardial infarction, stroke, ischaemia and pulmonary embolism
CC
XX
SQ
     Sequence 28 AA;
  Query Match
                          85.4%; Score 35; DB 4;
                                                     Length 28;
  Best Local Similarity
                          100.0%; Pred. No. 4.2;
             7; Conservative
                                                                      Gaps
                                                                               0;
  Matches
                                 0; Mismatches
                                                    0: Indels
                                                                  0;
Qy
            1 LVFFAED 7
              1111111
Dh
           17 LVFFAED 23
RESULT 92
AAB35596
     AAB35596 standard; peptide; 28 AA.
XX
AC
     AAB35596;
XX
DT
     15-FEB-2001 (first entry)
XX
     Human clone E11Q B(1-28) amyloid B peptide.
DE
XX
     Beta-amyloid; amyloid deposit; Alzheimer's disease; thrombolytic therapy;
KW
     acute cardiovascular disease; therapy.
KW
XX
OS
     Homo sapiens.
XX
     US6136548-A.
PN
XX
PD
     24-OCT-2000.
XX
                    99US-00388890.
PF
     02-SEP-1999;
XX
                    94US-00347144.
PR
     22-NOV-1994;
                    95WO-US015007.
PR
     22-NOV-1995;
PR
     26-JUL-1996;
                    96US-00686959.
XX
     (RUTF ) UNIV RUTGERS STATE NEW JERSEY.
PΑ
XX
```

```
PΙ
     Anderson S;
XX
DR
     WPI; 2001-030939/04.
XX
     Identifying mutant tissue-type plasminogen activator (t-PA) for improving
PT
     thrombolytic therapy or treating vascular hemorrhaging, by determining
PT
     whether t-PA binds to fibrin but not to a beta amyloid peptide.
PT
XX
     Example 3; Col 26; 23pp; English.
PS
XX
     The present invention describes a method for identifying mutant
CC
     derivatives of tissue-type plasminogen activator, which involves
CC
     determining whether or not they bind to beta-amyloid peptides and fibrin.
CC
     Mutants will only bind to the latter. These mutants are useful in
CC
     improved thrombolytic therapies, in the treatment of Alzheimer's disease
CC
     and in the treatment of acute cardiovascular disease, which may be caused
CC
     by myocardial infarction, stroke, ischaemia and pulmonary embolism
CC
XX
SQ
     Sequence 28 AA;
                          85.4%; Score 35; DB 4; Length 28;
  Query Match
                          100.0%; Pred. No. 4.2;
  Best Local Similarity
                                                                              0;
                                                                  0; Gaps
             7; Conservative
                               0; Mismatches
                                                   0; Indels
  Matches
            1 LVFFAED 7
Qу
              Db
           17 LVFFAED 23
RESULT 93
AAB35598
     AAB35598 standard; peptide; 28 AA.
ID
XX
AC
     AAB35598;
XX
     15-FEB-2001 (first entry)
DT
XX
     Human clone H14Q B(1-28) amyloid B peptide.
DE
XX
     Beta-amyloid; amyloid deposit; Alzheimer's disease; thrombolytic therapy;
KW
     acute cardiovascular disease; therapy.
KW
XX
OS
     Homo sapiens.
XX
PN
     US6136548-A.
XX
PD
     24-OCT-2000.
XX
                    99US-00388890.
PF
     02-SEP-1999;
XX
                    94US-00347144.
PR
     22-NOV-1994;
     22-NOV-1995;
                    95WO-US015007.
PR
                    96US-00686959.
PR
     26-JUL-1996;
XX
     (RUTF ) UNIV RUTGERS STATE NEW JERSEY.
PΑ
XX
PΙ
     Anderson S;
```

```
XX
DR
    WPI; 2001-030939/04.
XX
PT
     Identifying mutant tissue-type plasminogen activator (t-PA) for improving
PT
     thrombolytic therapy or treating vascular hemorrhaging, by determining
    whether t-PA binds to fibrin but not to a beta amyloid peptide.
PT
XX
PS
    Example 3; Col 26; 23pp; English.
XX
CC
    The present invention describes a method for identifying mutant
CC
    derivatives of tissue-type plasminogen activator, which involves
    determining whether or not they bind to beta-amyloid peptides and fibrin.
CC
    Mutants will only bind to the latter. These mutants are useful in
CC
CC
     improved thrombolytic therapies, in the treatment of Alzheimer's disease
CC
    and in the treatment of acute cardiovascular disease, which may be caused
CC
    by myocardial infarction, stroke, ischaemia and pulmonary embolism
XX
SO
     Sequence 28 AA;
  Query Match
                          85.4%; Score 35; DB 4; Length 28;
                          100.0%; Pred. No. 4.2;
 Best Local Similarity
            7; Conservative
                                0; Mismatches
                                                   0; Indels
                                                                  0; Gaps
                                                                              0;
            1 LVFFAED 7
Qу
              1111111
Db
           17 LVFFAED 23
RESULT 94
AAB35599
    AAB35599 standard; peptide; 28 AA.
XX
AC
    AAB35599;
XX
DT
    15-FEB-2001 (first entry)
XX
DE
    Human clone K16Q B(1-28) amyloid B peptide.
XX
     Beta-amyloid; amyloid deposit; Alzheimer's disease; thrombolytic therapy;
ΚW
KW
     acute cardiovascular disease; therapy.
XX
OS
    Homo sapiens.
XX
PN
    US6136548-A.
XX
PD
    24-OCT-2000.
XX
PF
     02-SEP-1999;
                    99US-00388890.
XX
                    94US-00347144.
PR
     22-NOV-1994;
PR
     22-NOV-1995;
                    95WO-US015007.
     26-JUL-1996;
                    96US-00686959.
PR
XX
PΑ
     (RUTF ) UNIV RUTGERS STATE NEW JERSEY.
XX
PΙ
    Anderson S;
XX
```

```
WPI; 2001-030939/04.
DR
XX
     Identifying mutant tissue-type plasminogen activator (t-PA) for improving
PT 
     thrombolytic therapy or treating vascular hemorrhaging, by determining
PT
     whether t-PA binds to fibrin but not to a beta amyloid peptide.
PT
XX
     Example 3; Col 26; 23pp; English.
PS
XX
     The present invention describes a method for identifying mutant
CC
     derivatives of tissue-type plasminogen activator, which involves
CC
     determining whether or not they bind to beta-amyloid peptides and fibrin.
CC
     Mutants will only bind to the latter. These mutants are useful in
CC
     improved thrombolytic therapies, in the treatment of Alzheimer's disease
CC
     and in the treatment of acute cardiovascular disease, which may be caused
CC
     by myocardial infarction, stroke, ischaemia and pulmonary embolism
CC
XX
     Sequence 28 AA;
SQ
                          85.4%; Score 35; DB 4; Length 28;
  Query Match
                          100.0%; Pred. No. 4.2;
  Best Local Similarity
                                                                              0;
             7; Conservative 0; Mismatches
                                                                  0; Gaps
                                                    0; Indels
  Matches
            1 LVFFAED 7
Qγ
              111111
           17 LVFFAED 23
Db
RESULT 95
AAB36202
     AAB36202 standard; peptide; 28 AA.
XX
     AAB36202;
AC
XX
     15-FEB-2001 (first entry)
DT
XX
     Human clone K28Q B(1-28) amyloid B peptide.
DE
XX
     Beta-amyloid; amyloid deposit; Alzheimer's disease; thrombolytic therapy;
KW
     acute cardiovascular disease; therapy.
KW
XX
OS
     Homo sapiens.
XX
PN
     US6136548-A.
XX
PD
     24-OCT-2000.
XX
     02-SEP-1999;
                    99US-00388890.
PF
XX
     22-NOV-1994;
                     94US-00347144.
PR
     22-NOV-1995;
                     95WO-US015007.
PR
                     96US-00686959.
PR
     26-JUL-1996;
XX
      (RUTF ) UNIV RUTGERS STATE NEW JERSEY.
PΑ
XX
PI
     Anderson S;
XX
     WPI; 2001-030939/04.
DR
```

```
XX
     Identifying mutant tissue-type plasminogen activator (t-PA) for improving
PT
     thrombolytic therapy or treating vascular hemorrhaging, by determining
PT
     whether t-PA binds to fibrin but not to a beta amyloid peptide.
PT
XX
     Example 3; Col 26; 23pp; English.
PS
XX
     The present invention describes a method for identifying mutant
CC
     derivatives of tissue-type plasminogen activator, which involves
CC
     determining whether or not they bind to beta-amyloid peptides and fibrin.
CC
     Mutants will only bind to the latter. These mutants are useful in
CC
     improved thrombolytic therapies, in the treatment of Alzheimer's disease
CC
     and in the treatment of acute cardiovascular disease, which may be caused
CC
     by myocardial infarction, stroke, ischaemia and pulmonary embolism
CC
XX
     Sequence 28 AA;
SO
                          85.4%; Score 35; DB 4; Length 28;
  Query Match
                          100.0%; Pred. No. 4.2;
  Best Local Similarity
                                                                  0; Gaps
                                                                              0;
                                                   0; Indels
                                 0; Mismatches
             7; Conservative
            1 LVFFAED 7
Qу
              17 LVFFAED 23
Db
RESULT 96
AAB35590
     AAB35590 standard; peptide; 28 AA.
XX
     AAB35590;
AC
XX
     15-FEB-2001 (first entry)
DΤ
XX
     Human clone B(1-28) amyloid B peptide.
DE
XX
     Beta-amyloid; amyloid deposit; Alzheimer's disease; thrombolytic therapy;
KW
     acute cardiovascular disease; therapy.
KW
XX
OS
     Homo sapiens.
XX
     US6136548-A.
ΡN
XX
PD
     24-OCT-2000.
XX
PF
     02-SEP-1999;
                    99US-00388890.
XX
                    94US-00347144.
     22-NOV-1994;
PR
PR
     22-NOV-1995;
                     95WO-US015007.
     26-JUL-1996;
                     96US-00686959.
PR
XX
      (RUTF ) UNIV RUTGERS STATE NEW JERSEY.
PΑ
XX
PI
     Anderson S;
XX
     WPI; 2001-030939/04.
DR
XX
```

```
Identifying mutant tissue-type plasminogen activator (t-PA) for improving
PT
     thrombolytic therapy or treating vascular hemorrhaging, by determining
PT
     whether t-PA binds to fibrin but not to a beta amyloid peptide.
PT
XX
     Example 3; Col 26; 23pp; English.
PS
XX
     The present invention describes a method for identifying mutant
CC
     derivatives of tissue-type plasminogen activator, which involves
CC
     determining whether or not they bind to beta-amyloid peptides and fibrin.
CC
     Mutants will only bind to the latter. These mutants are useful in
CC
     improved thrombolytic therapies, in the treatment of Alzheimer's disease
CC
     and in the treatment of acute cardiovascular disease, which may be caused
CC
     by myocardial infarction, stroke, ischaemia and pulmonary embolism
CC
XX
SO
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KW
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     Synthetic.
OS
ХX
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PN
XX
     23-NOV-2000.
PD
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     17-MAY-2000; 2000WO-US013576.
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 PR
      15-OCT-1999;
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 PR
XX
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      (CONJ-) CONJUCHEM INC.
XX
      Bridon DP, Ezrin AM, Milner PG, Holmes DL, Thibaudeau K;
 PΙ
XX
      WPI; 2001-112059/12.
 DR
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XX
     Modifying and attaching therapeutic peptides to albumin prevents
PT
     peptidase degradation, useful for increasing length of in vivo activity.
PT
XX
     Disclosure; Page 519; 733pp; English.
PS
XX
     The present invention describes a modified therapeutic peptide (I)
CC
     comprising a therapeutically active amino acid region (III) and a
CC
     reactive group (II) (e.g. succinimidyl and maleimido groups) attached to
CC
     a less therapeutically active amino acid region (IV), which covalently
CC
     bonds with amino/hydroxyl/thiol groups on blood components to form a
CC
     peptidase stabilised therapeutic peptide composed of 3-50 amino acids.
CC
     (I) are useful for modifying therapeutic peptides e.g. hormones, growth
CC
     factors and neurotransmitters, to protect them from peptidase activity in
CC
     vivo for the treatment of various disorders. Endogenous therapeutic
CC
     peptides are not suitable as drug candidates as they require frequent
CC
     administration due to rapid degradation by peptidases in the body.
CC
     Modifying and attaching therapeutic peptides to albumin prevents or
CC
     reduces the action of peptidases to increase length of activity (half
CC
     life) and specificity as bonding to large molecules decreases
CC
     intracellular uptake and interference with physiological processes.
CC
     AAB90829 to AAB92441 represent peptides which can be used in the
CC
     exemplification of the present invention
CC
XX
     Sequence 28 AA;
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                          85.4%; Score 35; DB 4; Length 28;
  Query Match
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  Matches
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Qу
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           17 LVFFAED 23
Db
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ידת
     22-JUN-2001
                  (first entry)
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     Protection; endogenous therapeutic peptide; peptidase; conjugation;
KW
     blood component; modification; succinimidyl; maleimido group; amino;
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     hydroxyl; thiol; hormone; growth factor; neurotransmitter.
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      17-MAY-2000; 2000WO-US013576.
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     15-OCT-1999;
                    99US-0159783P.
XX
PΑ
     (CONJ-) CONJUCHEM INC.
XX
     Bridon DP, Ezrin AM, Milner PG, Holmes DL, Thibaudeau K;
PΙ
XX
DR
     WPI; 2001-112059/12.
XX
     Modifying and attaching therapeutic peptides to albumin prevents
PT
     peptidase degradation, useful for increasing length of in vivo activity.
PT
XX
PS
     Disclosure; Page 509; 733pp; English.
XX
     The present invention describes a modified therapeutic peptide (I)
CC
     comprising a therapeutically active amino acid region (III) and a
CC
     reactive group (II) (e.g. succinimidyl and maleimido groups) attached to
CC
     a less therapeutically active amino acid region (IV), which covalently
CC
     bonds with amino/hydroxyl/thiol groups on blood components to form a
CC
     peptidase stabilised therapeutic peptide composed of 3-50 amino acids.
CC
     (I) are useful for modifying therapeutic peptides e.g. hormones, growth
CC
     factors and neurotransmitters, to protect them from peptidase activity in
CC
     vivo for the treatment of various disorders. Endogenous therapeutic
CC
     peptides are not suitable as drug candidates as they require frequent
CC
     administration due to rapid degradation by peptidases in the body.
CC
     Modifying and attaching therapeutic peptides to albumin prevents or
CC
     reduces the action of peptidases to increase length of activity (half
CC
     life) and specificity as bonding to large molecules decreases
CC
     intracellular uptake and interference with physiological processes.
CC
     AAB90829 to AAB92441 represent peptides which can be used in the
CC
CC
     exemplification of the present invention
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     22-JUN-2001
                 (first entry)
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KW
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hydroxyl; thiol; hormone; growth factor; neurotransmitter.
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OS
     Synthetic.
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     23-NOV-2000.
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PR
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                    99US-0153406P.
PR
                    99US-0159783P.
     15-OCT-1999;
PR
XX
     (CONJ-) CONJUCHEM INC.
PA
XX
PI
     Bridon DP, Ezrin AM, Milner PG, Holmes DL, Thibaudeau K;
XX
DR
     WPI; 2001-112059/12.
XX
PT
    Modifying and attaching therapeutic peptides to albumin prevents
     peptidase degradation, useful for increasing length of in vivo activity.
PT
XX
     Disclosure; Page 523; 733pp; English.
PS
XX
     The present invention describes a modified therapeutic peptide (I)
CC
     comprising a therapeutically active amino acid region (III) and a
CC
     reactive group (II) (e.g. succinimidyl and maleimido groups) attached to
CC
CC
     a less therapeutically active amino acid region (IV), which covalently
    bonds with amino/hydroxyl/thiol groups on blood components to form a
CC
     peptidase stabilised therapeutic peptide composed of 3-50 amino acids.
CC
     (I) are useful for modifying therapeutic peptides e.g. hormones, growth
CC
     factors and neurotransmitters, to protect them from peptidase activity in
CC
     vivo for the treatment of various disorders. Endogenous therapeutic
CC
     peptides are not suitable as drug candidates as they require frequent
CC
CC
     administration due to rapid degradation by peptidases in the body.
     Modifying and attaching therapeutic peptides to albumin prevents or
CC
CC
     reduces the action of peptidases to increase length of activity (half
     life) and specificity as bonding to large molecules decreases
CC
CC
     intracellular uptake and interference with physiological processes.
CC
     AAB90829 to AAB92441 represent peptides which can be used in the
CC
     exemplification of the present invention
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XX
PA
     (CONJ-) CONJUCHEM INC.
XX
PΙ
     Bridon DP, Ezrin AM, Milner PG, Holmes DL,
                                                    Thibaudeau K;
XX
DR
     WPI; 2001-112059/12.
XX
PT
     Modifying and attaching therapeutic peptides to albumin prevents
     peptidase degradation, useful for increasing length of in vivo activity.
PT
XX
PS
     Disclosure; Page 507; 733pp; English.
XX
CC
     The present invention describes a modified therapeutic peptide (I)
CC
    ·comprising a therapeutically active amino acid region (III) and a
CC
     reactive group (II) (e.g. succinimidyl and maleimido groups) attached to
     a less therapeutically active amino acid region (IV), which covalently
CC
CC
     bonds with amino/hydroxyl/thiol groups on blood components to form a
     peptidase stabilised therapeutic peptide composed of 3-50 amino acids.
CC
     (I) are useful for modifying therapeutic peptides e.g. hormones, growth
CC
CC
     factors and neurotransmitters, to protect them from peptidase activity in
CC
     vivo for the treatment of various disorders. Endogenous therapeutic
     peptides are not suitable as drug candidates as they require frequent
CC
CC
     administration due to rapid degradation by peptidases in the body.
CC
     Modifying and attaching therapeutic peptides to albumin prevents or
     reduces the action of peptidases to increase length of activity (half
CC
     life) and specificity as bonding to large molecules decreases
CC
     intracellular uptake and interference with physiological processes.
CC
CC
     AAB90829 to AAB92441 represent peptides which can be used in the
CC
     exemplification of the present invention
XX
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Search completed: February 28, 2004, 08:52:27 Job time: 100.5 secs

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OM protein - protein search, using sw model

Run on: February 28, 2004, 08:48:49; Search time 28.5 Seconds

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Perfect score: 41

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Scoring table: BLOSUM62

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Searched: 389414 seqs, 51625971 residues

Total number of hits satisfying chosen parameters: 389414

Minimum DB seq length: 0

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Post-processing: Minimum Match 0%

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Listing first 1000 summaries

Database : Issued Patents AA:*

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Pred. No. is the number of results predicted by chance to have a score greater than or equal to the score of the result being printed, and is derived by analysis of the total score distribution.

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35	85.4	12	2	US-08-986-948-11	Sequence 11, Appl
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987	24	58.5	629	4	US-09-107-532A-6656	Sequence	6656, Ap
988	24	58.5	637	1	US-08-235-838-14	Sequence	14, Appl
989	24	58.5	637	2	US-08-465-473B-14	Sequence	14, Appl
990	24	58.5	638	4	US-09-107-532A-3919	Sequence	3919, Ap
991	24	58.5	641	4	US-09-489-039A-12721	Sequence	12721, A
992	24	58.5	642	4	US-08-911-393-4	Sequence	4, Appli
993	24	58.5	673	4	US-09-091-725-13	Sequence	13, Appl
994	24	58.5	673	4	US-09-091-725-19	Sequence	19, Appl
995	24	58.5	673	4	US-09-091-725-23	Sequence	23, Appl
996	24	58.5	673	4	US-09-252-991A-28287	Sequence	28287, A
997	24	58.5	674	4	US-09-107-532A-6201	Sequence	6201, Ap
998	24	58.5	690	2	US-08-619-554-8	Sequence	8, Appli
999	24	58.5	696	4	US-09-907-794A-91	Sequence	91, Appl
1000	24	58.5	696	4	US-09-905-125A-91	Sequence	91, Appl

ALIGNMENTS

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RESULT 1
US-08-630-645-1
; Sequence 1, Application US/08630645
; Patent No. 5948763
  GENERAL INFORMATION:
    APPLICANT: SOTO-JARA, Claudio
    APPLICANT: BAUMANN, Marc
    APPLICANT: FRANGIONE, Blas
    TITLE OF INVENTION: PEPTIDES AND PHARMACEUTICAL COMPOSITIONS
    TITLE OF INVENTION: THEREOF FOR TREATMENT OF DISORDERS OR DISEASES
ASSOCIATED
    TITLE OF INVENTION: WITH PROTEIN FOLDING INTO AMYLOID OR AMYLOID-LIKE
DEPOSITS
    NUMBER OF SEQUENCES: 26
;
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: BROWDY AND NEIMARK
      STREET: 419 Seventh Street, N.W., Suite 400
      CITY: Washington
      STATE: D.C.
      COUNTRY: USA
      ZIP: 20004
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
       SOFTWARE: PatentIn Release #1.0, Version #1.30
     CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/630,645
      FILING DATE:
      CLASSIFICATION: 530
     PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/478,326
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FILING DATE: 06-JUN-1995
    ATTORNEY/AGENT INFORMATION:
      NAME: YUN, Allen C.
      REGISTRATION NUMBER: 37,971
      REFERENCE/DOCKET NUMBER: SOTO-JARA=1
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: 202-628-5197
      TELEFAX: 202-737-3528
  INFORMATION FOR SEQ ID NO: 1:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 8 amino acids
      TYPE: amino acid
      STRANDEDNESS: single
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
US-08-630-645-1
                         85.4%; Score 35; DB 2; Length 8;
 Query Match
 Best Local Similarity 100.0%; Pred. No. 3e+05;
         7; Conservative 0; Mismatches 0; Indels 0; Gaps
                                                                          0;
           1 LVFFAED 7
Qу
             2 LVFFAED 8
Db
RESULT 2
US-08-766-596A-1
; Sequence 1, Application US/08766596A
; Patent No. 6462171
; GENERAL INFORMATION:
    APPLICANT: SOTO-JARA, Claudio
    APPLICANT: BAUMANN, Marc
    APPLICANT: FRANGIONE, Blas
    TITLE OF INVENTION: PEPTIDES AND PHARMACEUTICAL
    TITLE OF INVENTION: COMPOSITIONS THEREOF FOR TREATMENT OF DISORDERS OR
DISEASES
    TITLE OF INVENTION: ASSOCIATED WITH PROTEIN FOLDING INTO AMYLOID OR
AMYLOID-LIKE
    TITLE OF INVENTION: DEPOSITS
    NUMBER OF SEQUENCES: 69
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: BROWDY AND NEIMARK
      STREET: 419 Seventh Street, N.W., Suite 400
      CITY: Washington
      STATE: D.C.
      COUNTRY: USA
;
      ZIP: 20004
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.30
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/766,596A
      FILING DATE:
      CLASSIFICATION: 435
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PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/630,645
      FILING DATE: 10-APR-1996
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/478,326
      FILING DATE: 06-JUN-1995
    ATTORNEY/AGENT INFORMATION:
      NAME: YUN, Allen C.
      REGISTRATION NUMBER: 37,971
      REFERENCE/DOCKET NUMBER: SOTO-JARA=1A
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: 202-628-5197
      TELEFAX: 202-737-3528
;
  INFORMATION FOR SEQ ID NO: 1:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 8 amino acids
      TYPE: amino acid
      STRANDEDNESS: single
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
US-08-766-596A-1
                         85.4%; Score 35; DB 4; Length 8;
 Query Match
 Best Local Similarity 100.0%; Pred. No. 3e+05;
         7; Conservative 0; Mismatches 0; Indels 0; Gaps
                                                                           0;
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Qу
             Db
           2 LVFFAED 8
RESULT 3
PCT-US96-10220-1
; Sequence 1, Application PC/TUS9610220
; GENERAL INFORMATION:
    APPLICANT:
    TITLE OF INVENTION: PEPTIDES AND PHARMACEUTICAL COMPOSITIONS
    TITLE OF INVENTION: THEREOF FOR TREATMENT OF DISORDERS OR DISEASES
ASSOCIATED
    TITLE OF INVENTION: WITH PROTEIN FOLDING INTO AMYLOID OR AMYLOID-LIKE
DEPOSITS
    NUMBER OF SEQUENCES: 26
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: BROWDY AND NEIMARK
      STREET: 419 Seventh Street, N.W., Suite 400
      CITY: Washington
      STATE: D.C.
      COUNTRY: USA
      ZIP: 20004
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.30
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: PCT/US96/10220
      FILING DATE:
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PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/478,326
      FILING DATE: 06-JUN-1995
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/630,645
      FILING DATE: 10-APR-1996
    ATTORNEY/AGENT INFORMATION:
      NAME: BROWDY, Roger L.
      REGISTRATION NUMBER: 25,618
      REFERENCE/DOCKET NUMBER: SOTO-JARA=1 PCT
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: 202-628-5197
      TELEFAX: 202-737-3528
  INFORMATION FOR SEQ ID NO: 1:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 8 amino acids
      TYPE: amino acid
      STRANDEDNESS: single
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
PCT-US96-10220-1
 Query Match
                         85.4%; Score 35; DB 5; Length 8;
 Best Local Similarity 100.0%; Pred. No. 3e+05;
 Matches
          7; Conservative 0; Mismatches 0; Indels
                                                               0; Gaps
Qу
           1 LVFFAED 7
             Dh
           2 LVFFAED 8
RESULT 4
US-08-766-596A-64
; Sequence 64, Application US/08766596A
; Patent No. 6462171
; GENERAL INFORMATION:
    APPLICANT: SOTO-JARA, Claudio
    APPLICANT: BAUMANN, Marc
    APPLICANT: FRANGIONE, Blas
    TITLE OF INVENTION: PEPTIDES AND PHARMACEUTICAL
    TITLE OF INVENTION: COMPOSITIONS THEREOF FOR TREATMENT OF DISORDERS OR
DISEASES
    TITLE OF INVENTION: ASSOCIATED WITH PROTEIN FOLDING INTO AMYLOID OR
AMYLOID-LIKE
    TITLE OF INVENTION: DEPOSITS
    NUMBER OF SEQUENCES: 69
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: BROWDY AND NEIMARK
      STREET: 419 Seventh Street, N.W., Suite 400
      CITY: Washington
      STATE: D.C.
      COUNTRY: USA
      ZIP: 20004
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
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SOFTWARE: PatentIn Release #1.0, Version #1.30
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/766,596A
      FILING DATE:
      CLASSIFICATION: 435
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/630,645
      FILING DATE: 10-APR-1996
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/478,326
      FILING DATE: 06-JUN-1995
    ATTORNEY/AGENT INFORMATION:
;
      NAME: YUN, Allen C.
      REGISTRATION NUMBER: 37,971
      REFERENCE/DOCKET NUMBER: SOTO-JARA=1A
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: 202-628-5197
      TELEFAX: 202-737-3528
   INFORMATION FOR SEQ ID NO: 64:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 9 amino acids
      TYPE: amino acid
;
      STRANDEDNESS: single
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
US-08-766-596A-64
  Query Match
                         85.4%; Score 35; DB 4; Length 9;
  Best Local Similarity 100.0%; Pred. No. 3e+05;
          7; Conservative 0; Mismatches 0; Indels
                                                                0; Gaps
           1 LVFFAED 7
Qу
             Db
           3 LVFFAED 9
RESULT 5
US-08-970-833-3
; Sequence 3, Application US/08970833
; Patent No. 6022859
  GENERAL INFORMATION:
    APPLICANT: Kiessling, Laura L.
    APPLICANT: Murphy, Regina M.
    TITLE OF INVENTION: INHIBITORS OF BETA-AMYLOID TOXICITY
    NUMBER OF SEQUENCES: 11
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: Quarles & Brady
;
      STREET: 411 East Wisconsin Avenue
      CITY: Milwaukee
      STATE: Wisconsin
      COUNTRY: U.S.A.
      ZIP: 53202-4497
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.25
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CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/970,833
      FILING DATE:
      CLASSIFICATION: 530
    ATTORNEY/AGENT INFORMATION:
      NAME: Baker, Jean C.
      REGISTRATION NUMBER: 35,433
      REFERENCE/DOCKET NUMBER: 960296.94291
    TELECOMMUNICATION INFORMATION:
       TELEPHONE: (414) 277-5709
       TELEFAX: (414) 271-3552
;
  INFORMATION FOR SEQ ID NO: 3:
;
    SEQUENCE CHARACTERISTICS:
;
      LENGTH: 10 amino acids
       TYPE: amino acid
       STRANDEDNESS: single
       TOPOLOGY: linear
    MOLECULE TYPE: peptide
US-08-970-833-3
 Query Match 85.4%; Score 35; DB 3; Length 10; Best Local Similarity 100.0%; Pred. No. 0.42;
          7; Conservative 0; Mismatches 0; Indels
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                                                                             0;
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Qу
             Db
           2 LVFFAED 8
RESULT 6
US-08-630-645-14
; Sequence 14, Application US/08630645
; Patent No. 5948763
 GENERAL INFORMATION:
    APPLICANT: SOTO-JARA, Claudio
    APPLICANT: BAUMANN, Marc
    APPLICANT: FRANGIONE, Blas
    TITLE OF INVENTION: PEPTIDES AND PHARMACEUTICAL COMPOSITIONS
     TITLE OF INVENTION: THEREOF FOR TREATMENT OF DISORDERS OR DISEASES
ASSOCIATED
     TITLE OF INVENTION: WITH PROTEIN FOLDING INTO AMYLOID OR AMYLOID-LIKE
DEPOSITS
    NUMBER OF SEQUENCES: 26
     CORRESPONDENCE ADDRESS:
       ADDRESSEE: BROWDY AND NEIMARK
;
       STREET: 419 Seventh Street, N.W., Suite 400
       CITY: Washington
;
       STATE: D.C.
       COUNTRY: USA
       ZIP: 20004
    COMPUTER READABLE FORM:
       MEDIUM TYPE: Floppy disk
       COMPUTER: IBM PC compatible
       OPERATING SYSTEM: PC-DOS/MS-DOS
       SOFTWARE: PatentIn Release #1.0, Version #1.30
     CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/630,645
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FILING DATE:
      CLASSIFICATION: 530
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/478,326
      FILING DATE: 06-JUN-1995
    ATTORNEY/AGENT INFORMATION:
     NAME: YUN, Allen C.
      REGISTRATION NUMBER: 37,971
      REFERENCE/DOCKET NUMBER: SOTO-JARA=1
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: 202-628-5197
      TELEFAX: 202-737-3528
  INFORMATION FOR SEQ ID NO:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 11 amino acids
      TYPE: amino acid
      STRANDEDNESS: single
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
US-08-630-645-14
  Query Match
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 Query Match 85.4%; Score 35; DB 2; Best Local Similarity 100.0%; Pred. No. 0.46;
  Matches 7; Conservative 0; Mismatches 0; Indels
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                                                                            0;
          1 LVFFAED 7
Qу
             3 LVFFAED 9
RESULT 7
US-08-766-596A-14
; Sequence 14, Application US/08766596A
; Patent No. 6462171
  GENERAL INFORMATION:
    APPLICANT: SOTO-JARA, Claudio
    APPLICANT: BAUMANN, Marc
    APPLICANT: FRANGIONE, Blas
    TITLE OF INVENTION: PEPTIDES AND PHARMACEUTICAL
    TITLE OF INVENTION: COMPOSITIONS THEREOF FOR TREATMENT OF DISORDERS OR
DISEASES
    TITLE OF INVENTION: ASSOCIATED WITH PROTEIN FOLDING INTO AMYLOID OR
AMYLOID-LIKE
    TITLE OF INVENTION: DEPOSITS
    NUMBER OF SEQUENCES: 69
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: BROWDY AND NEIMARK
      STREET: 419 Seventh Street, N.W., Suite 400
      CITY: Washington
      STATE: D.C.
      COUNTRY: USA
      ZIP: 20004
     COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.30
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CURRENT APPLICATION DATA:
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      FILING DATE:
      CLASSIFICATION: 435
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/630,645
      FILING DATE: 10-APR-1996
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/478,326
      FILING DATE: 06-JUN-1995
    ATTORNEY/AGENT INFORMATION:
      NAME: YUN, Allen C.
      REGISTRATION NUMBER: 37,971
      REFERENCE/DOCKET NUMBER: SOTO-JARA=1A
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: 202-628-5197
      TELEFAX: 202-737-3528
  INFORMATION FOR SEO ID NO: 14:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 11 amino acids
      TYPE: amino acid
      STRANDEDNESS: single
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
US-08-766-596A-14
 Query Match
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                                                              0; Gaps
                                                                          0;
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Qу
             3 LVFFAED 9
Dh
RESULT 8
PCT-US96-10220-14
; Sequence 14, Application PC/TUS9610220
  GENERAL INFORMATION:
    APPLICANT:
    TITLE OF INVENTION: PEPTIDES AND PHARMACEUTICAL COMPOSITIONS
    TITLE OF INVENTION: THEREOF FOR TREATMENT OF DISORDERS OR DISEASES
ASSOCIATED
    TITLE OF INVENTION: WITH PROTEIN FOLDING INTO AMYLOID OR AMYLOID-LIKE
DEPOSITS
    NUMBER OF SEQUENCES: 26
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: BROWDY AND NEIMARK
      STREET: 419 Seventh Street, N.W., Suite 400
      CITY: Washington
      STATE: D.C.
      COUNTRY: USA
      ZIP: 20004
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
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SOFTWARE: PatentIn Release #1.0, Version #1.30
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      APPLICATION NUMBER: PCT/US96/10220
      FILING DATE:
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/478,326
      FILING DATE: 06-JUN-1995
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/630,645
      FILING DATE: 10-APR-1996
    ATTORNEY/AGENT INFORMATION:
      NAME: BROWDY, Roger L.
      REGISTRATION NUMBER: 25,618
      REFERENCE/DOCKET NUMBER: SOTO-JARA=1 PCT
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: 202-628-5197
      TELEFAX: 202-737-3528
  INFORMATION FOR SEO ID NO: 14:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 11 amino acids
      TYPE: amino acid
      STRANDEDNESS: single
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
PCT-US96-10220-14
 Query Match
                         85.4%; Score 35; DB 5; Length 11;
 Best Local Similarity 100.0%; Pred. No. 0.46;
           7; Conservative 0; Mismatches 0; Indels 0; Gaps
 Matches
                                                                          0;
           1 LVFFAED 7
Qу
             3 LVFFAED 9
Db
RESULT 9
US-08-302-808-11
; Sequence 11, Application US/08302808
; Patent No. 5750349
  GENERAL INFORMATION:
    APPLICANT: SUZUKI, No. 5750349uhiro
    APPLICANT: ODAKA, Asano
    APPLICANT: KITADA, Chieko
    TITLE OF INVENTION: ANTIBODIES TO B-AMYLOIDS OR THEIR
    TITLE OF INVENTION: DERIVATIVES AND USE THEREOF
    NUMBER OF SEQUENCES: 14
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: DIKE, BRONSTEIN, ROBERTS & CUSHMAN
      STREET: 130 WATER STREET
      CITY: BOSTON
      STATE: MA
      COUNTRY: USA
      ZIP: 02019
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Diskette
      COMPUTER: IBM Compatible
      OPERATING SYSTEM: DOS
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SOFTWARE: FastSEQ Version 1.5
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/302,808
      FILING DATE: 15-SEP-1994
      CLASSIFICATION: 435
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: PCT/JP94/00089
      FILING DATE: 24-JAN-1994
      APPLICATION NUMBER: 010132/1993
      FILING DATE: 25-JAN-1993
      APPLICATION NUMBER: 019035/1993
      FILING DATE: 05-FEB-1993
      APPLICATION NUMBER: 286985/1993
      FILING DATE: 16-NOV-1993
      APPLICATION NUMBER: 334773/1993
      FILING DATE: 28-DEC-1993
    ATTORNEY/AGENT INFORMATION:
      NAME: DAVID, RESNICK S
      REGISTRATION NUMBER: 34,235
      REFERENCE/DOCKET NUMBER: 44631
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: 617-523-3400
      TELEFAX: 617-523-6440
      TELEX: 200291 STRE
   INFORMATION FOR SEQ ID NO:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 12 amino acids
      TYPE: amino acid
      STRANDEDNESS: single
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
    HYPOTHETICAL: NO
    ANTI-SENSE: NO
    FRAGMENT TYPE: N-terminal
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US-08-302-808-11
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 Matches
           7; Conservative 0; Mismatches 0; Indels
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           1 LVFFAED 7
Qy
             1 LVFFAED 7
RESULT 10
US-08-986-948-11
; Sequence 11, Application US/08986948
; Patent No. 5955317
   GENERAL INFORMATION:
    APPLICANT: SUZUKI, No. 5955317uhiro
    APPLICANT: ODAKA, Asano
    APPLICANT: KITADA, Chieko
    TITLE OF INVENTION: ANTIBODIES TO B-AMYLOIDS OR THEIR
    TITLE OF INVENTION: DERIVATIVES AND USE THEREOF
    NUMBER OF SEQUENCES: 14
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CORRESPONDENCE ADDRESS:
      ADDRESSEE: DIKE, BRONSTEIN, ROBERTS & CUSHMAN
      STREET: 130 WATER STREET
      CITY: BOSTON
      STATE: MA
      COUNTRY: USA
      ZIP: 02019
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Diskette
      COMPUTER: IBM Compatible
      OPERATING SYSTEM: DOS
      SOFTWARE: FastSEQ Version 1.5
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/986,948
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      FILING DATE:
      CLASSIFICATION:
    PRIOR APPLICATION DATA:
     APPLICATION NUMBER: 08/302,808
     FILING DATE: 15-SEP-1994
      APPLICATION NUMBER: PCT/JP94/00089
      FILING DATE: 24-JAN-1994
      APPLICATION NUMBER: 010132/1993
;
      FILING DATE: 25-JAN-1993
      APPLICATION NUMBER: 019035/1993
      FILING DATE: 05-FEB-1993
      APPLICATION NUMBER: 286985/1993
      FILING DATE: 16-NOV-1993
      APPLICATION NUMBER: 334773/1993
      FILING DATE: 28-DEC-1993
    ATTORNEY/AGENT INFORMATION:
      NAME: DAVID, RESNICK S
;
      REGISTRATION NUMBER: 34,235
      REFERENCE/DOCKET NUMBER: 44631
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: 617-523-3400
      TELEFAX: 617-523-6440
      TELEX: 200291 STRE
   INFORMATION FOR SEQ ID NO:
    SEQUENCE CHARACTERISTICS:
;
      LENGTH: 12 amino acids
;
      TYPE: amino acid
      STRANDEDNESS: single
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
    HYPOTHETICAL: NO
    ANTI-SENSE: NO
    FRAGMENT TYPE: N-terminal
    ORIGINAL SOURCE:
US-08-986-948-11
                         85.4%; Score 35; DB 2; Length 12;
 Query Match
 Best Local Similarity 100.0%; Pred. No. 0.51;
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           7; Conservative 0; Mismatches 0; Indels
 Matches
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Qу
           1 LVFFAED 7
             Db
           1 LVFFAED 7
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RESULT 11
US-09-458-481B-13
; Sequence 13, Application US/09458481B
; Patent No. 6310048
; GENERAL INFORMATION:
  APPLICANT: KUMAR, Vijaya B.
  TITLE OF INVENTION: ANTISENSE MODULATION OF AMYLOID BETA PROTEIN EXPRESSION
  FILE REFERENCE: 16153-9250
  CURRENT APPLICATION NUMBER: US/09/458,481B
  CURRENT FILING DATE: 1999-12-09
  NUMBER OF SEQ ID NOS: 20
  SOFTWARE: PatentIn Ver. 2.0
; SEQ ID NO 13
   LENGTH: 14
   TYPE: PRT
   ORGANISM: Homo sapiens
   FEATURE:
   OTHER INFORMATION: Description of Artificial Sequence: Amino Acids
    OTHER INFORMATION: Corresponding to Antisense Oligonucleotide
US-09-458-481B-13
  Query Match
                         85.4%; Score 35; DB 4; Length 14;
  Best Local Similarity 100.0%; Pred. No. 0.6;
            7; Conservative
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Qу
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             +111111
           1 LVFFAED 7
Db
RESULT 12
US-09-594-366-5
; Sequence 5, Application US/09594366
; Patent No. 6582945
; GENERAL INFORMATION:
  APPLICANT: Raso, Victor
  TITLE OF INVENTION: IMMUNOLOGICAL CONTROL OF BETA-AMYLOID LEVELS IN VIVO
 FILE REFERENCE: BBRI-2004
  CURRENT APPLICATION NUMBER: US/09/594,366
  CURRENT FILING DATE: 2000-06-15
  PRIOR APPLICATION NUMBER: 60/139,408
 PRIOR FILING DATE: 1999-06-16
 NUMBER OF SEQ ID NOS: 7
  SOFTWARE: PatentIn Ver. 2.0
; SEQ ID NO 5
   LENGTH: 14
   TYPE: PRT
    ORGANISM: Homo sapiens
US-09-594-366-5
                         85.4%; Score 35; DB 4; Length 14;
  Query Match
  Best Local Similarity 100.0%; Pred. No. 0.6;
            7; Conservative 0; Mismatches
 Matches
                                                  0; Indels
                                                                0; Gaps
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1 LVFFAED 7

Qу

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RESULT 13
US-08-612-785B-14
; Sequence 14, Application US/08612785B
; Patent No. 5854204
  GENERAL INFORMATION:
    APPLICANT: Findeis, Mark A. et al.
    TITLE OF INVENTION: Ab Peptides that Modulate b-Amyloid
    TITLE OF INVENTION: Aggregation
    NUMBER OF SEQUENCES:
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: LAHIVE & COCKFIELD
      STREET: 28 State Street, Suite 510
      CITY: Boston
      STATE: Massachusetts
      COUNTRY: USA
      ZIP: 02109-1875
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
;
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.25
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/612,785B
      FILING DATE: Herewith
      CLASSIFICATION: 514
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: USSN 08/404,831
;
      FILING DATE: 14-MAR-1995
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: USSN 08/475,579
                    07-JUN-1995
      FILING DATE:
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: USSN 08/548,998
      FILING DATE: 27-OCT-1995
    ATTORNEY/AGENT INFORMATION:
      NAME: DeConti, Giulio A.
      REGISTRATION NUMBER: 31,503
      REFERENCE/DOCKET NUMBER: PPI-002CP3
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: (617)227-7400
      TELEFAX: (617)742-4214
   INFORMATION FOR SEQ ID NO:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 15 amino acids
      TYPE: amino acid
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
     FRAGMENT TYPE: internal
US-08-612-785B-14
                         85.4%; Score 35; DB 2; Length 15;
  Query Match
  Best Local Similarity 100.0%; Pred. No. 0.65;
                                                                            0;
            7; Conservative 0; Mismatches
                                                  0; Indels
                                                                0; Gaps
  Matches
```

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Qy
          1 LVFFAED 7
              1111111
Dh
            2 LVFFAED 8
RESULT 14
US-08-612-785B-37
; Sequence 37, Application US/08612785B
; Patent No. 5854204
   GENERAL INFORMATION:
     APPLICANT: Findeis, Mark A. et al.
    TITLE OF INVENTION: Ab Peptides that Modulate b-Amyloid TITLE OF INVENTION: Aggregation
    NUMBER OF SEQUENCES: 40
    CORRESPONDENCE ADDRESS:
       ADDRESSEE: LAHIVE & COCKFIELD
       STREET: 28 State Street, Suite 510
       CITY: Boston
       STATE: Massachusetts
       COUNTRY: USA
       ZIP: 02109-1875
     COMPUTER READABLE FORM:
       MEDIUM TYPE: Floppy disk
       COMPUTER: IBM PC compatible
       OPERATING SYSTEM: PC-DOS/MS-DOS
       SOFTWARE: PatentIn Release #1.0, Version #1.25
     CURRENT APPLICATION DATA:
       APPLICATION NUMBER: US/08/612,785B
       FILING DATE: Herewith
       CLASSIFICATION: 514
    PRIOR APPLICATION DATA:
       APPLICATION NUMBER: USSN 08/404,831
       FILING DATE: 14-MAR-1995
     PRIOR APPLICATION DATA:
       APPLICATION NUMBER: USSN 08/475,579
       FILING DATE: 07-JUN-1995
     PRIOR APPLICATION DATA:
       APPLICATION NUMBER: USSN 08/548,998
       FILING DATE: 27-OCT-1995
     ATTORNEY/AGENT INFORMATION:
       NAME: DeConti, Giulio A.
       REGISTRATION NUMBER: 31,503
       REFERENCE/DOCKET NUMBER: PPI-002CP3
     TELECOMMUNICATION INFORMATION:
       TELEPHONE: (617)227-7400
       TELEFAX: (617)742-4214
   INFORMATION FOR SEQ ID NO:
     SEQUENCE CHARACTERISTICS:
       LENGTH: 15 amino acids
       TYPE: amino acid
       TOPOLOGY: linear
     MOLECULE TYPE: peptide
     FRAGMENT TYPE: internal
US-08-612-785B-37
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85.4%; Score 35; DB 2; Length 15;

Query Match

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Best Local Similarity 100.0%; Pred. No. 0.65;
  Matches
            7; Conservative 0; Mismatches 0; Indels
                                                                0; Gaps
                                                                            0;
            1 LVFFAED 7
Qу
              111111
            7 LVFFAED 13
RESULT 15
US-08-617-267C-14
; Sequence 14, Application US/08617267C
; Patent No. 6319498
   GENERAL INFORMATION:
     APPLICANT: Findeis, Mark A. et al.
    TITLE OF INVENTION: Modulators of Amyloid Aggregation
    NUMBER OF SEQUENCES: 45
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: LAHIVE & COCKFIELD, LLP
      STREET: 28 State Street
      CITY: Boston
      STATE: Massachusetts
      COUNTRY: USA
;
      ZIP: 02109-1875
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
       COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.25
     CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/617,267C
      FILING DATE: 14-MAR-1996
;
     PRIOR APPLICATION DATA:
      APPLICATION NUMBER: USSN 08/404,831
      FILING DATE: 14-MAR-1995
     PRIOR APPLICATION DATA:
      APPLICATION NUMBER: USSN 08/475,579
      FILING DATE: 07-JUN-1995
     PRIOR APPLICATION DATA:
;
      APPLICATION NUMBER: USSN 08/548,998
;
      FILING DATE: 27-OCT-1995
    ATTORNEY/AGENT INFORMATION:
      NAME: DeConti, Giulio A.
      REGISTRATION NUMBER: 31,503
      REFERENCE/DOCKET NUMBER: PPI-002CP2
     TELECOMMUNICATION INFORMATION:
      TELEPHONE: (617)227-7400
       TELEFAX: (617)227-5941
   INFORMATION FOR SEQ ID NO:
     SEQUENCE CHARACTERISTICS:
      LENGTH: 15 amino acids
      TYPE: amino acid
      TOPOLOGY: linear
     MOLECULE TYPE: peptide
     FRAGMENT TYPE: internal
US-08-617-267C-14
```

85.4%; Score 35; DB 4; Length 15;

Query Match

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Best Local Similarity 100.0%; Pred. No. 0.65;
 Matches
          7; Conservative 0; Mismatches 0; Indels 0; Gaps 0;
           1 LVFFAED 7
Qу
             2 LVFFAED 8
RESULT 16
US-08-766-596A-56
; Sequence 56, Application US/08766596A
; Patent No. 6462171
  GENERAL INFORMATION:
    APPLICANT: SOTO-JARA, Claudio
    APPLICANT: BAUMANN, Marc
    APPLICANT: FRANGIONE, Blas
    TITLE OF INVENTION: PEPTIDES AND PHARMACEUTICAL
    TITLE OF INVENTION: COMPOSITIONS THEREOF FOR TREATMENT OF DISORDERS OR
DISEASES
    TITLE OF INVENTION: ASSOCIATED WITH PROTEIN FOLDING INTO AMYLOID OR
AMYLOID-LIKE
    TITLE OF INVENTION: DEPOSITS
;
    NUMBER OF SEQUENCES: 69
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: BROWDY AND NEIMARK
      STREET: 419 Seventh Street, N.W., Suite 400
      CITY: Washington
      STATE: D.C.
      COUNTRY: USA
      ZIP: 20004
    COMPUTER READABLE FORM:
;
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.30
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/766,596A
;
      FILING DATE:
      CLASSIFICATION: 435
;
    PRIOR APPLICATION DATA:
;
      APPLICATION NUMBER: US 08/630,645
      FILING DATE: 10-APR-1996
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/478,326
      FILING DATE: 06-JUN-1995
    ATTORNEY/AGENT INFORMATION:
;
      NAME: YUN, Allen C.
;
      REGISTRATION NUMBER: 37,971
ï
      REFERENCE/DOCKET NUMBER: SOTO-JARA=1A
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: 202-628-5197
      TELEFAX: 202-737-3528
  INFORMATION FOR SEQ ID NO:
    SEQUENCE CHARACTERISTICS:
;
      LENGTH: 15 amino acids
;
      TYPE: amino acid
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STRANDEDNESS: single

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TOPOLOGY: linear
     MOLECULE TYPE: peptide
US-08-766-596A-56
  Query Match 85.4%; Score 35; DB 4; Length 15; Best Local Similarity 100.0%; Pred. No. 0.65;
            7; Conservative
                               0; Mismatches 0; Indels
                                                                0; Gaps
                                                                             0;
           1 LVFFAED 7
Qу
              6 LVFFAED 12
RESULT 17
US-08-766-596A-57
; Sequence 57, Application US/08766596A
; Patent No. 6462171
  GENERAL INFORMATION:
     APPLICANT: SOTO-JARA, Claudio
    APPLICANT: BAUMANN, Marc
;
     APPLICANT: FRANGIONE, Blas
     TITLE OF INVENTION: PEPTIDES AND PHARMACEUTICAL
     TITLE OF INVENTION: COMPOSITIONS THEREOF FOR TREATMENT OF DISORDERS OR
DISEASES
     TITLE OF INVENTION: ASSOCIATED WITH PROTEIN FOLDING INTO AMYLOID OR
AMYLOID-LIKE
     TITLE OF INVENTION: DEPOSITS
     NUMBER OF SEQUENCES: 69
     CORRESPONDENCE ADDRESS:
;
       ADDRESSEE: BROWDY AND NEIMARK
       STREET: 419 Seventh Street, N.W., Suite 400
      CITY: Washington
       STATE: D.C.
      COUNTRY: USA
      ZIP: 20004
     COMPUTER READABLE FORM:
       MEDIUM TYPE: Floppy disk
       COMPUTER: IBM PC compatible
;
       OPERATING SYSTEM: PC-DOS/MS-DOS
       SOFTWARE: PatentIn Release #1.0, Version #1.30
     CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/766,596A
       FILING DATE:
      CLASSIFICATION: 435
     PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/630,645
;
       FILING DATE: 10-APR-1996
     PRIOR APPLICATION DATA:
       APPLICATION NUMBER: US 08/478,326
       FILING DATE: 06-JUN-1995
     ATTORNEY/AGENT INFORMATION:
       NAME: YUN, Allen C.
       REGISTRATION NUMBER: 37,971
      REFERENCE/DOCKET NUMBER: SOTO-JARA=1A
     TELECOMMUNICATION INFORMATION:
;
      TELEPHONE: 202-628-5197
       TELEFAX: 202-737-3528
```

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INFORMATION FOR SEQ ID NO:
     SEQUENCE CHARACTERISTICS:
      LENGTH: 15 amino acids
      TYPE: amino acid
      STRANDEDNESS: single
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
US-08-766-596A-57
  Query Match
                         85.4%; Score 35; DB 4; Length 15;
  Best Local Similarity 100.0%; Pred. No. 0.65;
 Matches 7; Conservative 0; Mismatches
                                                 0; Indels
                                                               0; Gaps
                                                                           0;
           1 LVFFAED 7
Qу
             Db
           6 LVFFAED 12
RESULT 18
US-08-766-596A-58
; Sequence 58, Application US/08766596A
; Patent No. 6462171
  GENERAL INFORMATION:
    APPLICANT: SOTO-JARA, Claudio
    APPLICANT: BAUMANN, Marc
    APPLICANT: FRANGIONE, Blas
    TITLE OF INVENTION: PEPTIDES AND PHARMACEUTICAL
    TITLE OF INVENTION: COMPOSITIONS THEREOF FOR TREATMENT OF DISORDERS OR
DISEASES
    TITLE OF INVENTION: ASSOCIATED WITH PROTEIN FOLDING INTO AMYLOID OR
AMYLOID-LIKE
    TITLE OF INVENTION: DEPOSITS
    NUMBER OF SEQUENCES: 69
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: BROWDY AND NEIMARK
      STREET: 419 Seventh Street, N.W., Suite 400
      CITY: Washington
      STATE: D.C.
      COUNTRY: USA
;
      ZIP: 20004
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.30
;
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/766,596A
;
      FILING DATE:
;
      CLASSIFICATION: 435
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/630,645
      FILING DATE: 10-APR-1996
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/478,326
      FILING DATE: 06-JUN-1995
;
    ATTORNEY/AGENT INFORMATION:
      NAME: YUN, Allen C.
```

```
REGISTRATION NUMBER: 37,971
       REFERENCE/DOCKET NUMBER: SOTO-JARA=1A
    TELECOMMUNICATION INFORMATION:
       TELEPHONE: 202-628-5197
       TELEFAX: 202-737-3528
   INFORMATION FOR SEQ ID NO: 58:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 15 amino acids
      TYPE: amino acid
      STRANDEDNESS: single
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
US-08-766-596A-58
                         85.4%; Score 35; DB 4; Length 15;
  Query Match
  Best Local Similarity 100.0%; Pred. No. 0.65;
           7; Conservative 0; Mismatches 0; Indels
                                                                0; Gaps
                                                                            0;
           1 LVFFAED 7
Qy
              111111
Db
           6 LVFFAED 12
RESULT 19
US-08-766-596A-59
; Sequence 59, Application US/08766596A
; Patent No. 6462171
  GENERAL INFORMATION:
    APPLICANT: SOTO-JARA, Claudio
    APPLICANT: BAUMANN, Marc
    APPLICANT: FRANGIONE, Blas
    TITLE OF INVENTION: PEPTIDES AND PHARMACEUTICAL
    TITLE OF INVENTION: COMPOSITIONS THEREOF FOR TREATMENT OF DISORDERS OR
DISEASES
    TITLE OF INVENTION: ASSOCIATED WITH PROTEIN FOLDING INTO AMYLOID OR
AMYLOID-LIKE
    TITLE OF INVENTION: DEPOSITS
    NUMBER OF SEQUENCES: 69
;
    CORRESPONDENCE ADDRESS:
;
      ADDRESSEE: BROWDY AND NEIMARK
      STREET: 419 Seventh Street, N.W., Suite 400
      CITY: Washington
      STATE: D.C.
      COUNTRY: USA
;
      ZIP: 20004
;
    COMPUTER READABLE FORM:
;
      MEDIUM TYPE: Floppy disk
;
      COMPUTER: IBM PC compatible
;
      OPERATING SYSTEM: PC-DOS/MS-DOS
       SOFTWARE: PatentIn Release #1.0, Version #1.30
     CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/766,596A
      FILING DATE:
       CLASSIFICATION: 435
     PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/630,645
      FILING DATE: 10-APR-1996
```

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PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/478,326
      FILING DATE: 06-JUN-1995
    ATTORNEY/AGENT INFORMATION:
      NAME: YUN, Allen C.
      REGISTRATION NUMBER: 37,971
      REFERENCE/DOCKET NUMBER: SOTO-JARA=1A
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: 202-628-5197
      TELEFAX: 202-737-3528
  INFORMATION FOR SEQ ID NO:
                              59:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 15 amino acids
      TYPE: amino acid
      STRANDEDNESS: single
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
US-08-766-596A-59
                         85.4%; Score 35; DB 4; Length 15;
  Query Match
 Best Local Similarity 100.0%; Pred. No. 0.65;
            7; Conservative
                               0; Mismatches
                                                  0; Indels
                                                                0; Gaps
                                                                            0;
           1 LVFFAED 7
Qу
             111111
           6 LVFFAED 12
RESULT 20
US-08-766-596A-63
; Sequence 63, Application US/08766596A
; Patent No. 6462171
  GENERAL INFORMATION:
    APPLICANT: SOTO-JARA, Claudio
    APPLICANT: BAUMANN, Marc
    APPLICANT: FRANGIONE, Blas
    TITLE OF INVENTION: PEPTIDES AND PHARMACEUTICAL
    TITLE OF INVENTION: COMPOSITIONS THEREOF FOR TREATMENT OF DISORDERS OR
DISEASES
    TITLE OF INVENTION: ASSOCIATED WITH PROTEIN FOLDING INTO AMYLOID OR
AMYLOID-LIKE
    TITLE OF INVENTION: DEPOSITS
;
    NUMBER OF SEQUENCES: 69
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: BROWDY AND NEIMARK
      STREET: 419 Seventh Street, N.W., Suite 400
      CITY: Washington
;
      STATE: D.C.
      COUNTRY: USA
      ZIP: 20004
;
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.30
;
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/766,596A
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FILING DATE:
      CLASSIFICATION: 435
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/630,645
      FILING DATE: 10-APR-1996
    PRIOR APPLICATION DATA:
;
      APPLICATION NUMBER: US 08/478,326
      FILING DATE: 06-JUN-1995
    ATTORNEY/AGENT INFORMATION:
      NAME: YUN, Allen C.
      REGISTRATION NUMBER: 37,971
      REFERENCE/DOCKET NUMBER: SOTO-JARA=1A
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: 202-628-5197
      TELEFAX: 202-737-3528
  INFORMATION FOR SEQ ID NO:
                             63:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 15 amino acids
      TYPE: amino acid
      STRANDEDNESS: single
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
US-08-766-596A-63
 Query Match
                         85.4%; Score 35; DB 4; Length 15;
 Best Local Similarity 100.0%; Pred. No. 0.65;
           7; Conservative 0; Mismatches
                                                0; Indels
                                                               0; Gaps
                                                                           0;
           1 LVFFAED 7
             6 LVFFAED 12
RESULT 21
US-08-766-596A-65
; Sequence 65, Application US/08766596A
; Patent No. 6462171
 GENERAL INFORMATION:
    APPLICANT: SOTO-JARA, Claudio
    APPLICANT: BAUMANN, Marc
    APPLICANT: FRANGIONE, Blas
    TITLE OF INVENTION: PEPTIDES AND PHARMACEUTICAL
    TITLE OF INVENTION: COMPOSITIONS THEREOF FOR TREATMENT OF DISORDERS OR
DISEASES
    TITLE OF INVENTION: ASSOCIATED WITH PROTEIN FOLDING INTO AMYLOID OR
AMYLOID-LIKE
    TITLE OF INVENTION: DEPOSITS
    NUMBER OF SEQUENCES: 69
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: BROWDY AND NEIMARK
      STREET: 419 Seventh Street, N.W., Suite 400
      CITY: Washington
      STATE: D.C.
      COUNTRY: USA
      ZIP: 20004
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
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COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.30
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/766,596A
      FILING DATE:
      CLASSIFICATION: 435
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/630,645
      FILING DATE: 10-APR-1996
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: US 08/478,326
      FILING DATE: 06-JUN-1995
;
    ATTORNEY/AGENT INFORMATION:
;
      NAME: YUN, Allen C.
      REGISTRATION NUMBER: 37,971
      REFERENCE/DOCKET NUMBER: SOTO-JARA=1A
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: 202-628-5197
      TELEFAX: 202-737-3528
   INFORMATION FOR SEQ ID NO:
;
    SEQUENCE CHARACTERISTICS:
      LENGTH: 15 amino acids
;
      TYPE: amino acid
      STRANDEDNESS: single
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
US-08-766-596A-65
  Query Match
                         85.4%; Score 35; DB 4; Length 15;
  Best Local Similarity
                         100.0%; Pred. No. 0.65;
 Matches
            7; Conservative
                              0; Mismatches
                                                  0; Indels
                                                                 0; Gaps
                                                                             0;
            1 LVFFAED 7
Qу
              111111
            6 LVFFAED 12
RESULT 22
US-09-264-709A-2
; Sequence 2, Application US/09264709A
; Patent No. 6320024
; GENERAL INFORMATION:
  APPLICANT: Roberts, Eugene
  TITLE OF INVENTION: Method for Design of Substances that Enhance Memory and
  TITLE OF INVENTION: Improve the Quality of Life
  FILE REFERENCE: 2124-310
  CURRENT APPLICATION NUMBER: US/09/264,709A
  CURRENT FILING DATE: 1999-03-09
  PRIOR APPLICATION NUMBER: 08/797,782
  PRIOR FILING DATE: 1997-02-07
  NUMBER OF SEQ ID NOS: 39
  SOFTWARE: PatentIn Ver. 2.1
 SEQ ID NO 2
;
   LENGTH: 17
;
   TYPE: PRT
   ORGANISM: Homo sapiens
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US-09-264-709A-2
 Query Match
                         85.4%; Score 35; DB 4; Length 17;
 Best Local Similarity 100.0%; Pred. No. 0.74;
 Matches
           7; Conservative
                               0; Mismatches
                                               0; Indels
                                                               0; Gaps
                                                                           0;
           1 LVFFAED 7
Qу
             6 LVFFAED 12
Db
RESULT 23
US-09-594-366-3
; Sequence 3, Application US/09594366
; Patent No. 6582945
; GENERAL INFORMATION:
; APPLICANT: Raso, Victor
  TITLE OF INVENTION: IMMUNOLOGICAL CONTROL OF BETA-AMYLOID LEVELS IN VIVO
 FILE REFERENCE: BBRI-2004
  CURRENT APPLICATION NUMBER: US/09/594,366
  CURRENT FILING DATE: 2000-06-15
  PRIOR APPLICATION NUMBER: 60/139,408
 PRIOR FILING DATE: 1999-06-16
;
 NUMBER OF SEQ ID NOS: 7
  SOFTWARE: PatentIn Ver. 2.0
; SEQ ID NO 3
   LENGTH: 17
   TYPE: PRT
   ORGANISM: Homo sapiens
US-09-594-366-3
 Query Match
                         85.4%; Score 35; DB 4; Length 17;
 Best Local Similarity 100.0%; Pred. No. 0.74;
            7; Conservative 0; Mismatches 0; Indels
 Matches
                                                                   Gaps
                                                                           0;
           1 LVFFAED 7
Qу
             111111
           9 LVFFAED 15
Db
RESULT 24
US-08-970-833-11
; Sequence 11, Application US/08970833
; Patent No. 6022859
  GENERAL INFORMATION:
    APPLICANT: Kiessling, Laura L.
    APPLICANT: Murphy, Regina M.
;
    TITLE OF INVENTION: INHIBITORS OF BETA-AMYLOID TOXICITY
;
    NUMBER OF SEQUENCES: 11
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: Quarles & Brady
      STREET: 411 East Wisconsin Avenue
```

CITY: Milwaukee STATE: Wisconsin COUNTRY: U.S.A.

ZIP: 53202-4497 COMPUTER READABLE FORM:

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MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.25
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/970,833
      FILING DATE:
      CLASSIFICATION: 530
    ATTORNEY/AGENT INFORMATION:
      NAME: Baker, Jean C.
      REGISTRATION NUMBER: 35,433
;
      REFERENCE/DOCKET NUMBER: 960296.94291
    TELECOMMUNICATION INFORMATION:
;
      TELEPHONE: (414) 277-5709
      TELEFAX: (414) 271-3552
  INFORMATION FOR SEQ ID NO: 11:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 19 amino acids
      TYPE: amino acid
      STRANDEDNESS: single
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
US-08-970-833-11
 Query Match
                         85.4%; Score 35; DB 3; Length 19;
 Best Local Similarity 100.0%; Pred. No. 0.83;
 Matches
           7; Conservative 0; Mismatches 0; Indels
                                                               0; Gaps
Qу
           1 LVFFAED 7
             11 LVFFAED 17
RESULT 25
US-08-970-833-10
; Sequence 10, Application US/08970833
; Patent No. 6022859
  GENERAL INFORMATION:
    APPLICANT: Kiessling, Laura L.
    APPLICANT: Murphy, Regina M.
    TITLE OF INVENTION: INHIBITORS OF BETA-AMYLOID TOXICITY
    NUMBER OF SEQUENCES: 11
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: Quarles & Brady
      STREET: 411 East Wisconsin Avenue
      CITY: Milwaukee
      STATE: Wisconsin
      COUNTRY: U.S.A.
;
      ZIP: 53202-4497
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.25
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/970,833
      FILING DATE:
```

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CLASSIFICATION: 530
    ATTORNEY/AGENT INFORMATION:
      NAME: Baker, Jean C.
      REGISTRATION NUMBER: 35,433
      REFERENCE/DOCKET NUMBER: 960296.94291
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: (414) 277-5709
      TELEFAX: (414) 271-3552
  INFORMATION FOR SEQ ID NO: 10:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 20 amino acids
;
      TYPE: amino acid
;
      STRANDEDNESS: single
;
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
    FEATURE:
      NAME/KEY: Peptide
      LOCATION: 13..14
      OTHER INFORMATION: /note= "amino caproate should
      OTHER INFORMATION: appear between residues 13 and 14."
US-08-970-833-10
                         85.4%; Score 35; DB 3; Length 20;
  Query Match
  Best Local Similarity 100.0%; Pred. No. 0.88;
           7; Conservative 0; Mismatches
                                                  0; Indels
                                                                0; Gaps
                                                                           0;
           1 LVFFAED 7
Qу
             Db
           4 LVFFAED 10
RESULT 26
US-08-304-585-7
; Sequence 7, Application US/08304585
; Patent No. 5721106
  GENERAL INFORMATION:
     APPLICANT: Maggio, John E.
    APPLICANT: Mantyh, Patrick W.
    TITLE OF INVENTION: LABELLED BETA-AMYLOID PEPTIDE AND
    TITLE OF INVENTION: METHODS FOR USE IN DETECTING ALZHEIMER'S DISEASE
    NUMBER OF SEQUENCES: 12
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: Mueting, Raasch, Gebhardt & Schwappach, P.A.
      STREET: P.O. Box 581415
      CITY: Minneapolis
      STATE: MN
    COUNTRY: USA
      ZIP: 55458-1415
     COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
       SOFTWARE: PatentIn Release #1.0, Version #1.30
     CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/304,585
      FILING DATE: 12-SEP-1994
      CLASSIFICATION: 435
```

```
ATTORNEY/AGENT INFORMATION:
      NAME: Mueting, Ann M.
      REGISTRATION NUMBER: 33,977
      REFERENCE/DOCKET NUMBER: 110.00010120
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: 612-305-1217
      TELEFAX: 612-305-1228
   INFORMATION FOR SEQ ID NO:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 26 amino acids
      TYPE: amino acid
      STRANDEDNESS: not relevant
      TOPOLOGY: not relevant
    MOLECULE TYPE: peptide
US-08-304-585-7
 Query Match
                         85.4%; Score 35; DB 1; Length 26;
 Best Local Similarity 100.0%; Pred. No. 1.2;
            7; Conservative 0; Mismatches 0; Indels
                                                               0; Gaps
                                                                           0;
 Matches
Qу
           1 LVFFAED 7
             8 LVFFAED 14
RESULT 27
US-08-346-849-4
; Sequence 4, Application US/08346849
; Patent No. 5670483
  GENERAL INFORMATION:
    APPLICANT: Zhang, Shuguang
    APPLICANT: Lockshin, Curtis
    APPLICANT: Rich, Alexander
    APPLICANT: Holmes, Todd
    TITLE OF INVENTION: STABLE MACROSCOPIC MEMBRANES FORMED BY
    TITLE OF INVENTION: SELF-ASSEMBLY OF AMPHIPHILIC PEPTIDES AND USES
    TITLE OF INVENTION: THEREFOR
    NUMBER OF SEQUENCES: 64
    CORRESPONDENCE ADDRESS:
;
      ADDRESSEE: HAMILTON, BROOK, SMITH & REYNOLDS, P.C.
      STREET: Two Militia Drive
      CITY: Lexington
      STATE: Massachusetts
      COUNTRY: U.S.A.
      ZIP: 02173-4799
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
;
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.25
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/346,849
      FILING DATE:
      CLASSIFICATION: 435
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: 07/973,326
      FILING DATE: 28 DECEMBER 1992
```

```
ATTORNEY/AGENT INFORMATION:
      NAME: Brook, David E.
       REGISTRATION NUMBER: 22,592
      REFERENCE/DOCKET NUMBER: MIT-6008
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: (617) 861-6240
       TELEFAX: (617) 861-9540
   INFORMATION FOR SEQ ID NO: 4:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 28 amino acids
;
      TYPE: amino acid
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
US-08-346-849-4
  Query Match
                         85.4%; Score 35; DB 1; Length 28;
  Best Local Similarity
                         100.0%; Pred. No. 1.3;
            7; Conservative
                             0; Mismatches 0; Indels
                                                                0; Gaps
                                                                            0;
           1 LVFFAED 7
Qу
              1111111
          17 LVFFAED 23
RESULT 28
US-08-302-808-7
; Sequence 7, Application US/08302808
; Patent No. 5750349
   GENERAL INFORMATION:
    APPLICANT: SUZUKI, No. 5750349uhiro
    APPLICANT: ODAKA, Asano
;
    APPLICANT: KITADA, Chieko
    TITLE OF INVENTION: ANTIBODIES TO B-AMYLOIDS OR THEIR
    TITLE OF INVENTION: DERIVATIVES AND USE THEREOF
    NUMBER OF SEQUENCES: 14
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: DIKE, BRONSTEIN, ROBERTS & CUSHMAN
      STREET: 130 WATER STREET
      CITY: BOSTON
      STATE: MA
;
      COUNTRY: USA
      ZIP: 02019
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Diskette
      COMPUTER: IBM Compatible
      OPERATING SYSTEM: DOS
      SOFTWARE: FastSEQ Version 1.5
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/302,808
      FILING DATE: 15-SEP-1994
      CLASSIFICATION: 435
     PRIOR APPLICATION DATA:
      APPLICATION NUMBER: PCT/JP94/00089
      FILING DATE: 24-JAN-1994
      APPLICATION NUMBER: 010132/1993
;
      FILING DATE: 25-JAN-1993
      APPLICATION NUMBER: 019035/1993
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FILING DATE: 05-FEB-1993
      APPLICATION NUMBER: 286985/1993
      FILING DATE: 16-NOV-1993
      APPLICATION NUMBER: 334773/1993
      FILING DATE: 28-DEC-1993
    ATTORNEY/AGENT INFORMATION:
      NAME: DAVID, RESNICK S
      REGISTRATION NUMBER: 34,235
      REFERENCE/DOCKET NUMBER: 44631
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: 617-523-3400
      TELEFAX: 617-523-6440
      TELEX: 200291 STRE
;
  INFORMATION FOR SEQ ID NO:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 28 amino acids
      TYPE: amino acid
      STRANDEDNESS: single
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
    HYPOTHETICAL: NO
    ANTI-SENSE: NO
    FRAGMENT TYPE: N-terminal
    ORIGINAL SOURCE:
US-08-302-808-7
 Query Match
                         85.4%; Score 35; DB 1; Length 28;
                         100.0%; Pred. No. 1.3;
 Best Local Similarity
 Matches
            7; Conservative 0; Mismatches
                                                  0; Indels
                                                                0; Gaps
                                                                            0;
           1 LVFFAED 7
Qу
             111111
Db
          17 LVFFAED 23
RESULT 29
US-08-609-090-2
; Sequence 2, Application US/08609090
; Patent No. 5840838
  GENERAL INFORMATION:
    APPLICANT: HENSLEY, Kenneth
    APPLICANT: BUTTERFIELD, D. A.
    APPLICANT: CARNEY, John M.
    APPLICANT: AKSENOV, Michael
    TITLE OF INVENTION: A PROCESS FOR ENHANCING THE ACTIVITY OF
    TITLE OF INVENTION: AN OLIGOPEPTIDE OR POLYPEPTIDES
    NUMBER OF SEQUENCES: 11
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: LOWE PRICE LEBLANC & BECKER
      STREET: 99 Canal Center Plaza, Suite 300
      CITY: Alexandria
      STATE: Virginia
      COUNTRY: USA
      ZIP: 22314
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
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OPERATING SYSTEM: PC-DOS/MS-DOS
       SOFTWARE: PatentIn Release #1.0, Version #1.30
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/609,090
      FILING DATE: 29-FEB-1996
      CLASSIFICATION: 530
    ATTORNEY/AGENT INFORMATION:
      NAME: Kraus, Eric J.
      REGISTRATION NUMBER: 36,190
      REFERENCE/DOCKET NUMBER: 434-059
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: 703-684-1111
      TELEFAX: 703-684-1124
  INFORMATION FOR SEQ ID NO:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 28 amino acids
      TYPE: amino acid
      STRANDEDNESS: single
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
US-08-609-090-2
 Query Match
                         85.4%; Score 35; DB 2; Length 28;
 Best Local Similarity 100.0%; Pred. No. 1.3;
 Matches
           7; Conservative 0; Mismatches
                                                  0; Indels
                                                                0; Gaps
Qу
           1 LVFFAED 7
             1111111
Db
          17 LVFFAED 23
RESULT 30
US-08-986-948-7
; Sequence 7, Application US/08986948
; Patent No. 5955317
  GENERAL INFORMATION:
    APPLICANT: SUZUKI, No. 5955317uhiro
    APPLICANT: ODAKA, Asano
    APPLICANT: KITADA, Chieko
    TITLE OF INVENTION: ANTIBODIES TO B-AMYLOIDS OR THEIR
    TITLE OF INVENTION: DERIVATIVES AND USE THEREOF
    NUMBER OF SEQUENCES: 14
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: DIKE, BRONSTEIN, ROBERTS & CUSHMAN
      STREET: 130 WATER STREET
      CITY: BOSTON
       STATE: MA
      COUNTRY: USA
;
      ZIP: 02019
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Diskette
      COMPUTER: IBM Compatible
      OPERATING SYSTEM: DOS
      SOFTWARE: FastSEQ Version 1.5
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/986,948
      FILING DATE:
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CLASSIFICATION:
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: 08/302,808
      FILING DATE: 15-SEP-1994
      APPLICATION NUMBER: PCT/JP94/00089
      FILING DATE: 24-JAN-1994
      APPLICATION NUMBER: 010132/1993
      FILING DATE: 25-JAN-1993
      APPLICATION NUMBER: 019035/1993
      FILING DATE: 05-FEB-1993
      APPLICATION NUMBER: 286985/1993
      FILING DATE: 16-NOV-1993
      APPLICATION NUMBER: 334773/1993
      FILING DATE: 28-DEC-1993
    ATTORNEY/AGENT INFORMATION:
      NAME: DAVID, RESNICK S
      REGISTRATION NUMBER: 34,235
      REFERENCE/DOCKET NUMBER: 44631
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: 617-523-3400
      TELEFAX: 617-523-6440
      TELEX: 200291 STRE
   INFORMATION FOR SEQ ID NO:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 28 amino acids
      TYPE: amino acid
      STRANDEDNESS: single
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
    HYPOTHETICAL: NO
    ANTI-SENSE: NO
    FRAGMENT TYPE: N-terminal
  ORIGINAL SOURCE:
US-08-986-948-7
 Query Match
                         85.4%; Score 35; DB 2; Length 28;
 Best Local Similarity 100.0%; Pred. No. 1.3;
 Matches 7; Conservative 0; Mismatches 0; Indels
                                                               0; Gaps
           1 LVFFAED 7
Qу
             111111
Db
          17 LVFFAED 23
RESULT 31
US-08-293-284A-4
; Sequence 4, Application US/08293284A
; Patent No. 5955343
  GENERAL INFORMATION:
    APPLICANT: Holmes, Todd
    APPLICANT: Zhang, Shuguang
    APPLICANT: Rich, Alexander
                DiPersio, C. Michael
    APPLICANT:
    APPLICANT: Lockshin, Curtis
    TITLE OF INVENTION: STABLE MACROSCOPIC MEMBRANES FORMED BY
    TITLE OF INVENTION: SELF-ASSEMBLY OF AMPHIPHILIC PEPTIDES AND USES
    TITLE OF INVENTION: THEREFOR
```

```
NUMBER OF SEQUENCES:
                          64
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: HAMILTON, BROOK, SMITH & REYNOLDS, P.C.
      STREET: Two Militia Drive
      CITY: Lexington
      STATE: Massachusetts
      COUNTRY: U.S.A.
      ZIP: 02173-4799
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.25
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/293,284A
      FILING DATE: 22-AUG-1994
;
      CLASSIFICATION: 435
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: 07/973,326
      FILING DATE: 28-DEC-1992
    ATTORNEY/AGENT INFORMATION:
      NAME: Brook, David E.
;
      REGISTRATION NUMBER: 22,592
      REFERENCE/DOCKET NUMBER: MIT-6008A
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: (617) 861-6240
      TELEFAX: (617) 861-9540
  INFORMATION FOR SEQ ID NO: 4:
     SEQUENCE CHARACTERISTICS:
      LENGTH: 28 amino acids
      TYPE: amino acid
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
US-08-293-284A-4
  Query Match
                         85.4%; Score 35; DB 2; Length 28;
  Best Local Similarity 100.0%; Pred. No. 1.3;
            7; Conservative 0; Mismatches
                                                                           0;
                                                  0; Indels
                                                                0; Gaps
 Matches
           1 LVFFAED 7
Qу
             17 LVFFAED 23
Db
RESULT 32
US-08-461-216-2
; Sequence 2, Application US/08461216
; Patent No. 5958883
  GENERAL INFORMATION:
     APPLICANT: Snow, A.D.
     TITLE OF INVENTION: ANIMAL MODELS OF HUMAN AMYLOIDOSES
    NUMBER OF SEQUENCES: 8
     CORRESPONDENCE ADDRESS:
       ADDRESSEE: Christensen, O'Connor, Johnson and Kindness
       STREET: 1420 Fifth Avenue, Suite 2800
      CITY: Seattle
       STATE: Washington
```

```
COUNTRY: USA
      ZIP: 98101-2347
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Diskette-5.25 inch, 1.2Mb storage
      COMPUTER: IBM PC/386 Compatible
      OPERATING SYSTEM: MS-DOS 4.01
      SOFTWARE: Word for Windows-t
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/08/461,216
      FILING DATE:
      CLASSIFICATION:
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: 07/969,734
      FILING DATE: October 23, 1992
      APPLICATION NUMBER: 07/950,417
      FILING DATE: September 23, 1992
    ATTORNEY/AGENT INFORMATION:
      NAME: Broderick, Thomas F.
      REGISTRATION NUMBER: 31,332
      REFERENCE/DOCKET NUMBER: UOFW-1-6707
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: 1-206-682-8100; 1-206-224-0709 (direct)
      TELEFAX: 1-206-224-0779
      TELEX: 4938023
  INFORMATION FOR SEQ ID NO:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 28 amino acids
      TYPE: amino acid
      STRANDEDNESS: single
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
      DESCRIPTION: {SYMBOL 98 \f "Symbol"}/A4(1-28);
      DESCRIPTION: page 83, line 31
US-08-461-216-2
                         85.4%; Score 35; DB 2; Length 28;
  Query Match
  Best Local Similarity 100.0%; Pred. No. 1.3;
            7; Conservative 0; Mismatches
                                                  0; Indels
                                                                0; Gaps
                                                                           0;
 Matches
           1 LVFFAED 7
Qу
             Db
          17 LVFFAED 23
RESULT 33
US-09-388-890-2
; Sequence 2, Application US/09388890
; Patent No. 6136548
   GENERAL INFORMATION:
    APPLICANT: ANDERSON, STEPHEN
    TITLE OF INVENTION: METHODS FOR THE PREVENTION AND TREATMENT
    TITLE OF INVENTION: OF VASCULAR HEMORRHAGING AND ALZHEIMER'S DISEASE
    NUMBER OF SEQUENCES: 14
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: HOWREY & SIMON
      STREET: 1299 PENNSYLVANIA AVENUE, N.W.
      CITY: WASHINGTON
```

```
STATE: D.C.
      COUNTRY: US
      ZIP: 20004
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.25
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/09/388,890
      FILING DATE:
      CLASSIFICATION:
;
    PRIOR APPLICATION DATA:
;
      APPLICATION NUMBER: 08/686,959
      FILING DATE:
    ATTORNEY/AGENT INFORMATION:
      NAME: AUERBACH, JEFFREY I.
      REGISTRATION NUMBER: 32,680
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: (202) 383-7451
      TELEFAX: (202) 383-6610
  INFORMATION FOR SEQ ID NO: 2:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 28 amino acids
      TYPE: amino acid
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
    HYPOTHETICAL: NO
    FRAGMENT TYPE: N-terminal
    ORIGINAL SOURCE:
      ORGANISM: HOMO SAPIENS
    IMMEDIATE SOURCE:
      CLONE: B(1-28) peptide of amyloid B protein
US-09-388-890-2
  Query Match
                         85.4%; Score 35; DB 3; Length 28;
 Best Local Similarity 100.0%; Pred. No. 1.3;
           7; Conservative 0; Mismatches 0; Indels
                                                               0; Gaps
           1 LVFFAED 7
Qy
             Db
         17 LVFFAED 23
RESULT 34
US-09-388-890-3
; Sequence 3, Application US/09388890
; Patent No. 6136548
  GENERAL INFORMATION:
    APPLICANT: ANDERSON, STEPHEN
    TITLE OF INVENTION: METHODS FOR THE PREVENTION AND TREATMENT
    TITLE OF INVENTION: OF VASCULAR HEMORRHAGING AND ALZHEIMER'S DISEASE
    NUMBER OF SEQUENCES: 14
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: HOWREY & SIMON
      STREET: 1299 PENNSYLVANIA AVENUE, N.W.
      CITY: WASHINGTON
```

```
STATE: D.C.
      COUNTRY: US
      ZIP: 20004
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.25
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/09/388,890
      FILING DATE:
      CLASSIFICATION:
;
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: 08/686,959
      FILING DATE:
    ATTORNEY/AGENT INFORMATION:
      NAME: AUERBACH, JEFFREY I.
      REGISTRATION NUMBER: 32,680
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: (202) 383-7451
      TELEFAX: (202) 383-6610
  INFORMATION FOR SEQ ID NO: 3:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 28 amino acids
      TYPE: amino acid
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
    HYPOTHETICAL: YES
    FRAGMENT TYPE: N-terminal
    ORIGINAL SOURCE:
      ORGANISM: HOMO SAPIENS
    IMMEDIATE SOURCE:
      CLONE: D1N B(1-28) peptide of amyloid B protein
US-09-388-890-3
 Query Match
                         85.4%; Score 35; DB 3; Length 28;
 Best Local Similarity 100.0%; Pred. No. 1.3;
            7; Conservative 0; Mismatches 0; Indels
                                                               0; Gaps
                                                                           0;
           1 LVFFAED 7
Qу
             Db
          17 LVFFAED 23
RESULT 35
US-09-388-890-4
; Sequence 4, Application US/09388890
; Patent No. 6136548
  GENERAL INFORMATION:
    APPLICANT: ANDERSON, STEPHEN
    TITLE OF INVENTION: METHODS FOR THE PREVENTION AND TREATMENT
    TITLE OF INVENTION: OF VASCULAR HEMORRHAGING AND ALZHEIMER'S DISEASE
    NUMBER OF SEQUENCES: 14
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: HOWREY & SIMON
      STREET: 1299 PENNSYLVANIA AVENUE, N.W.
      CITY: WASHINGTON
```

```
STATE: D.C.
      COUNTRY: US
      ZIP: 20004
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.25
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/09/388,890
      FILING DATE:
      CLASSIFICATION:
;
    PRIOR APPLICATION DATA:
;
      APPLICATION NUMBER: 08/686,959
      FILING DATE:
    ATTORNEY/AGENT INFORMATION:
      NAME: AUERBACH, JEFFREY I.
      REGISTRATION NUMBER: 32,680
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: (202) 383-7451
      TELEFAX: (202) 383-6610
;
   INFORMATION FOR SEQ ID NO: 4:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 28 amino acids
      TYPE: amino acid
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
    HYPOTHETICAL: YES
    FRAGMENT TYPE: N-terminal
;
    ORIGINAL SOURCE:
      ORGANISM: HOMO SAPIENS
     IMMEDIATE SOURCE:
      CLONE: E3Q B(1-28) peptide of amyloid B protein
US-09-388-890-4
                         85.4%; Score 35; DB 3; Length 28;
  Query Match
  Best Local Similarity 100.0%; Pred. No. 1.3;
 Matches 7; Conservative 0; Mismatches 0; Indels
                                                               0; Gaps
           1 LVFFAED 7
Qу
             111111
          17 LVFFAED 23
RESULT 36
US-09-388-890-5
; Sequence 5, Application US/09388890
; Patent No. 6136548
   GENERAL INFORMATION:
    APPLICANT: ANDERSON, STEPHEN
    TITLE OF INVENTION: METHODS FOR THE PREVENTION AND TREATMENT
    TITLE OF INVENTION: OF VASCULAR HEMORRHAGING AND ALZHEIMER'S DISEASE
    NUMBER OF SEQUENCES: 14
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: HOWREY & SIMON
      STREET: 1299 PENNSYLVANIA AVENUE, N.W.
      CITY: WASHINGTON
```

```
STATE: D.C.
      COUNTRY: US
      ZIP: 20004
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.25
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/09/388,890
      FILING DATE:
      CLASSIFICATION:
;
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: 08/686,959
      FILING DATE:
    ATTORNEY/AGENT INFORMATION:
      NAME: AUERBACH, JEFFREY I.
      REGISTRATION NUMBER: 32,680
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: (202) 383-7451
      TELEFAX: (202) 383-6610
   INFORMATION FOR SEQ ID NO: 5:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 28 amino acids
      TYPE: amino acid
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
;
    HYPOTHETICAL: YES
    FRAGMENT TYPE: N-terminal
;
    ORIGINAL SOURCE:
      ORGANISM: HOMO SAPIENS
     IMMEDIATE SOURCE:
      CLONE: R5Q B(1-28) peptide of amyloid B protein
US-09-388-890-5
                         85.4%; Score 35; DB 3; Length 28;
  Query Match
  Best Local Similarity 100.0%; Pred. No. 1.3;
           7; Conservative 0; Mismatches
                                                                0; Gaps
                                                                            0;
                                                  0; Indels
           1 LVFFAED 7
Qу
             +1111111
          17 LVFFAED 23
RESULT 37
US-09-388-890-6
; Sequence 6, Application US/09388890
; Patent No. 6136548
   GENERAL INFORMATION:
     APPLICANT: ANDERSON, STEPHEN
     TITLE OF INVENTION: METHODS FOR THE PREVENTION AND TREATMENT
    TITLE OF INVENTION: OF VASCULAR HEMORRHAGING AND ALZHEIMER'S DISEASE
    NUMBER OF SEQUENCES: 14
     CORRESPONDENCE ADDRESS:
     ADDRESSEE: HOWREY & SIMON
      STREET: 1299 PENNSYLVANIA AVENUE, N.W.
      CITY: WASHINGTON
```

```
STATE: D.C.
      COUNTRY: US
      ZIP: 20004
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.25
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/09/388,890
      FILING DATE:
      CLASSIFICATION:
    PRIOR APPLICATION DATA:
;
      APPLICATION NUMBER: 08/686,959
      FILING DATE:
    ATTORNEY/AGENT INFORMATION:
      NAME: AUERBACH, JEFFREY I.
      REGISTRATION NUMBER: 32,680
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: (202) 383-7451
      TELEFAX: (202) 383-6610
  INFORMATION FOR SEQ ID NO: 6:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 28 amino acids
      TYPE: amino acid
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
    HYPOTHETICAL: YES
    FRAGMENT TYPE: N-terminal
    ORIGINAL SOURCE:
      ORGANISM: HOMO SAPIENS
    IMMEDIATE SOURCE:
      CLONE: H6Q B(1-28) peptide of amyloid B protein
US-09-388-890-6
                         85.4%; Score 35; DB 3; Length 28;
 Query Match
 Best Local Similarity 100.0%; Pred. No. 1.3;
           7; Conservative 0; Mismatches 0; Indels
                                                               0; Gaps
                                                                           0;
           1 LVFFAED 7
Qy
             17 LVFFAED 23
Db
RESULT 38
US-09-388-890-7
; Sequence 7, Application US/09388890
; Patent No. 6136548
  GENERAL INFORMATION:
    APPLICANT: ANDERSON, STEPHEN
    TITLE OF INVENTION: METHODS FOR THE PREVENTION AND TREATMENT
    TITLE OF INVENTION: OF VASCULAR HEMORRHAGING AND ALZHEIMER'S DISEASE
    NUMBER OF SEQUENCES: 14
    CORRESPONDENCE ADDRESS:
     ADDRESSEE: HOWREY & SIMON
      STREET: 1299 PENNSYLVANIA AVENUE, N.W.
      CITY: WASHINGTON
```

```
STATE: D.C.
      COUNTRY: US
      ZIP: 20004
     COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.25
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/09/388,890
      FILING DATE:
      CLASSIFICATION:
    PRIOR APPLICATION DATA:
;
      APPLICATION NUMBER: 08/686,959
      FILING DATE:
    ATTORNEY/AGENT INFORMATION:
      NAME: AUERBACH, JEFFREY I.
      REGISTRATION NUMBER: 32,680
     TELECOMMUNICATION INFORMATION:
      TELEPHONE: (202) 383-7451
      TELEFAX: (202) 383-6610
;
   INFORMATION FOR SEQ ID NO: 7:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 28 amino acids
      TYPE: amino acid
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
    HYPOTHETICAL: YES
    FRAGMENT TYPE: N-terminal
    ORIGINAL SOURCE:
      ORGANISM: HOMO SAPIENS
     IMMEDIATE SOURCE:
      CLONE: D7Q B(1-28) peptide of amyloid B protein
US-09-388-890-7
                         85.4%; Score 35; DB 3; Length 28;
  Query Match
  Best Local Similarity 100.0%; Pred. No. 1.3;
            7; Conservative 0; Mismatches 0; Indels
                                                                0; Gaps
                                                                            0;
           1 LVFFAED 7
Qy
             111111
Db
          17 LVFFAED 23
RESULT 39
US-09-388-890-8
; Sequence 8, Application US/09388890
; Patent No. 6136548
   GENERAL INFORMATION:
     APPLICANT: ANDERSON, STEPHEN
     TITLE OF INVENTION: METHODS FOR THE PREVENTION AND TREATMENT
     TITLE OF INVENTION: OF VASCULAR HEMORRHAGING AND ALZHEIMER'S DISEASE
     NUMBER OF SEQUENCES: 14
     CORRESPONDENCE ADDRESS:
      ADDRESSEE: HOWREY & SIMON
      STREET: 1299 PENNSYLVANIA AVENUE, N.W.
      CITY: WASHINGTON
```

```
STATE: D.C.
      COUNTRY: US
      ZIP: 20004
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.25
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/09/388,890
      FILING DATE:
      CLASSIFICATION:
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: 08/686,959
      FILING DATE:
    ATTORNEY/AGENT INFORMATION:
      NAME: AUERBACH, JEFFREY I.
      REGISTRATION NUMBER: 32,680
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: (202) 383-7451
      TELEFAX: (202) 383-6610
   INFORMATION FOR SEQ ID NO: 8:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 28 amino acids
      TYPE: amino acid
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
    HYPOTHETICAL: YES
    FRAGMENT TYPE: N-terminal
    ORIGINAL SOURCE:
      ORGANISM: HOMO SAPIENS
    IMMEDIATE SOURCE:
      CLONE: E11Q B(1-28) peptide of amyloid B protein
US-09-388-890-8
                         85.4%; Score 35; DB 3; Length 28;
 Query Match
 Best Local Similarity 100.0%; Pred. No. 1.3;
            7; Conservative 0; Mismatches 0; Indels
                                                                            0;
                                                                0; Gaps
           1 LVFFAED 7
Qу
             Db
          17 LVFFAED 23
RESULT 40
US-09-388-890-9
; Sequence 9, Application US/09388890
; Patent No. 6136548
   GENERAL INFORMATION:
    APPLICANT: ANDERSON, STEPHEN
    TITLE OF INVENTION: METHODS FOR THE PREVENTION AND TREATMENT
    TITLE OF INVENTION: OF VASCULAR HEMORRHAGING AND ALZHEIMER'S DISEASE
    NUMBER OF SEQUENCES: 14
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: HOWREY & SIMON
      STREET: 1299 PENNSYLVANIA AVENUE, N.W.
      CITY: WASHINGTON
```

```
STATE: D.C.
      COUNTRY: US
      ZIP: 20004
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.25
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/09/388,890
      FILING DATE:
      CLASSIFICATION:
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: 08/686,959
;
      FILING DATE:
    ATTORNEY/AGENT INFORMATION:
      NAME: AUERBACH, JEFFREY I.
      REGISTRATION NUMBER: 32,680
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: (202) 383-7451
      TELEFAX: (202) 383-6610
   INFORMATION FOR SEQ ID NO: 9:
;
    SEQUENCE CHARACTERISTICS:
      LENGTH: 28 amino acids
      TYPE: amino acid
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
    HYPOTHETICAL: YES
    FRAGMENT TYPE: N-terminal
    ORIGINAL SOURCE:
      ORGANISM: HOMO SAPIENS
    IMMEDIATE SOURCE:
      CLONE: H13Q B(1-28) peptide of amyloid B protein
US-09-388-890-9
  Query Match
                         85.4%; Score 35; DB 3; Length 28;
  Best Local Similarity 100.0%; Pred. No. 1.3;
 Matches 7; Conservative 0; Mismatches 0; Indels
                                                               0; Gaps
                                                                           0;
           1 LVFFAED 7
Qу
             111111
Db
          17 LVFFAED 23
RESULT 41
US-09-388-890-10
; Sequence 10, Application US/09388890
; Patent No. 6136548
  GENERAL INFORMATION:
    APPLICANT: ANDERSON, STEPHEN
    TITLE OF INVENTION: METHODS FOR THE PREVENTION AND TREATMENT
    TITLE OF INVENTION: OF VASCULAR HEMORRHAGING AND ALZHEIMER'S DISEASE
    NUMBER OF SEQUENCES: 14
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: HOWREY & SIMON
      STREET: 1299 PENNSYLVANIA AVENUE, N.W.
      CITY: WASHINGTON
```

```
STATE: D.C.
       COUNTRY: US
       ZIP: 20004
     COMPUTER READABLE FORM:
       MEDIUM TYPE: Floppy disk
       COMPUTER: IBM PC compatible
       OPERATING SYSTEM: PC-DOS/MS-DOS
       SOFTWARE: PatentIn Release #1.0, Version #1.25
     CURRENT APPLICATION DATA:
       APPLICATION NUMBER: US/09/388,890
       FILING DATE:
      CLASSIFICATION:
     PRIOR APPLICATION DATA:
;
      APPLICATION NUMBER: 08/686,959
;
       FILING DATE:
     ATTORNEY/AGENT INFORMATION:
      NAME: AUERBACH, JEFFREY I.
       REGISTRATION NUMBER: 32,680
     TELECOMMUNICATION INFORMATION:
       TELEPHONE: (202) 383-7451
       TELEFAX: (202) 383-6610
   INFORMATION FOR SEQ ID NO: 10:
     SEQUENCE CHARACTERISTICS:
;
       LENGTH: 28 amino acids
       TYPE: amino acid
       TOPOLOGY: linear
    MOLECULE TYPE: peptide
    HYPOTHETICAL: YES
     FRAGMENT TYPE: N-terminal
     ORIGINAL SOURCE:
      ORGANISM: HOMO SAPIENS
     IMMEDIATE SOURCE:
      CLONE: H14Q B(1-28) peptide of amyloid B protein
US-09-388-890-10
  Query Match
                         85.4%; Score 35; DB 3; Length 28;
  Best Local Similarity 100.0%; Pred. No. 1.3;
            7; Conservative 0; Mismatches 0; Indels
  Matches
                                                                0; Gaps
                                                                            0;
            1 LVFFAED 7
Qу
             Dh
          17 LVFFAED 23
RESULT 42
US-09-388-890-11
; Sequence 11, Application US/09388890
; Patent No. 6136548
   GENERAL INFORMATION:
     APPLICANT: ANDERSON, STEPHEN
     TITLE OF INVENTION: METHODS FOR THE PREVENTION AND TREATMENT
     TITLE OF INVENTION: OF VASCULAR HEMORRHAGING AND ALZHEIMER'S DISEASE
    NUMBER OF SEQUENCES: 14
     CORRESPONDENCE ADDRESS:
      ADDRESSEE: HOWREY & SIMON
       STREET: 1299 PENNSYLVANIA AVENUE, N.W.
      CITY: WASHINGTON
```

```
STATE: D.C.
      COUNTRY: US
      ZIP: 20004
     COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.25
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/09/388,890
     FILING DATE:
;
     CLASSIFICATION:
    PRIOR APPLICATION DATA:
;
      APPLICATION NUMBER: 08/686,959
      FILING DATE:
    ATTORNEY/AGENT INFORMATION:
      NAME: AUERBACH, JEFFREY I.
      REGISTRATION NUMBER: 32,680
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: (202) 383-7451
      TELEFAX: (202) 383-6610
   INFORMATION FOR SEQ ID NO: 11:
    SEQUENCE CHARACTERISTICS:
      LENGTH: 28 amino acids
      TYPE: amino acid
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
    HYPOTHETICAL: YES
    FRAGMENT TYPE: N-terminal
    ORIGINAL SOURCE:
      ORGANISM: HOMO SAPIENS
     IMMEDIATE SOURCE:
      CLONE: K16Q B(1-28) peptide of amyloid B protein
US-09-388-890-11
  Query Match
                         85.4%; Score 35; DB 3; Length 28;
  Best Local Similarity 100.0%; Pred. No. 1.3;
          7; Conservative 0; Mismatches 0; Indels
 Matches
                                                               0; Gaps
                                                                           0;
           1 LVFFAED 7
Qу
             17 LVFFAED 23
RESULT 43
US-09-388-890-14
; Sequence 14, Application US/09388890
; Patent No. 6136548
; GENERAL INFORMATION:
    APPLICANT: ANDERSON, STEPHEN
    TITLE OF INVENTION: METHODS FOR THE PREVENTION AND TREATMENT
    TITLE OF INVENTION: OF VASCULAR HEMORRHAGING AND ALZHEIMER'S DISEASE
    NUMBER OF SEQUENCES: 14
    CORRESPONDENCE ADDRESS:
      ADDRESSEE: HOWREY & SIMON
      STREET: 1299 PENNSYLVANIA AVENUE, N.W.
      CITY: WASHINGTON
```

```
STATE: D.C.
      COUNTRY: US
      ZIP: 20004
    COMPUTER READABLE FORM:
      MEDIUM TYPE: Floppy disk
      COMPUTER: IBM PC compatible
      OPERATING SYSTEM: PC-DOS/MS-DOS
      SOFTWARE: PatentIn Release #1.0, Version #1.25
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER: US/09/388,890
      FILING DATE:
      CLASSIFICATION:
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER: 08/686,959
      FILING DATE:
    ATTORNEY/AGENT INFORMATION:
      NAME: AUERBACH, JEFFREY I.
      REGISTRATION NUMBER:
    TELECOMMUNICATION INFORMATION:
      TELEPHONE: (202) 383-7451
      TELEFAX: (202) 383-6610
   INFORMATION FOR SEQ ID NO: 14:
    SEOUENCE CHARACTERISTICS:
      LENGTH: 28 amino acids
      TYPE: amino acid
      TOPOLOGY: linear
    MOLECULE TYPE: peptide
    HYPOTHETICAL: YES
    FRAGMENT TYPE: N-terminal
    ORIGINAL SOURCE:
      ORGANISM: HOMO SAPIENS
    IMMEDIATE SOURCE:
      CLONE: K28Q B(1-28) peptide of amyloid B protein
US-09-388-890-14
 Query Match
                         85.4%; Score 35; DB 3; Length 28;
                         100.0%; Pred. No. 1.3;
 Best Local Similarity
            7; Conservative
                               0; Mismatches
 Matches
                                                  0; Indels
                                                                0; Gaps
                                                                            0;
           1 LVFFAED 7
Qу
             Dh
          17 LVFFAED 23
RESULT 44
US-09-264-709A-1
; Sequence 1, Application US/09264709A
; Patent No. 6320024
; GENERAL INFORMATION:
; APPLICANT: Roberts, Eugene
  TITLE OF INVENTION: Method for Design of Substances that Enhance Memory and
                       Improve the Quality of Life
  TITLE OF INVENTION:
                   2124-310
 FILE REFERENCE:
  CURRENT APPLICATION NUMBER: US/09/264,709A
; CURRENT FILING DATE: 1999-03-09
; PRIOR APPLICATION NUMBER: 08/797,782
; PRIOR FILING DATE: 1997-02-07
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NUMBER OF SEQ ID NOS: 39
  SOFTWARE: PatentIn Ver. 2.1
; SEQ ID NO 1
    LENGTH: 28
    TYPE: PRT
    ORGANISM: Homo sapiens
US-09-264-709A-1
                          85.4%; Score 35; DB 4; Length 28;
  Query Match
  Best Local Similarity
                          100.0%; Pred. No. 1.3;
  Matches
             7; Conservative
                                 0; Mismatches
                                                   0;
                                                       Indels
                                                                  0; Gaps
                                                                              0;
            1 LVFFAED 7
Qу
              Db
           17 LVFFAED 23
RESULT 45
US-08-723-661B-2
; Sequence 2, Application US/08723661B
 Patent No. 6340783
    GENERAL INFORMATION:
         APPLICANT: Alan D Snow
         TITLE OF INVENTION: Animal Models of Human Amyloidoses
         NUMBER OF SEQUENCES: 7
         CORRESPONDENCE ADDRESS:
              ADDRESSEE: Patrick M. Dwyer
              STREET: 1818 Westlake Avenue N, Suite 114
              CITY: Seattle
              STATE: WA (Washington)
              COUNTRY: United States of America
              ZIP: 98109
         COMPUTER READABLE FORM:
              MEDIUM TYPE: Diskette - 3.50 inch, 1.44 Mb storage
              COMPUTER: IBM PC
              OPERATING SYSTEM: PC-DOS (Windows 98)
              SOFTWARE: WordPerfect 5.2
         CURRENT APPLICATION DATA:
              APPLICATION NUMBER: US/08/723,661B
              FILING DATE: 31-Oct-1996
         PRIOR APPLICATION DATA:
              APPLICATION NUMBER: 08/461,216
              FILING DATE: 05-Jun-1995
              APPLICATION NUMBER: 07/969,734
              FILING DATE: 23-Oct-1992
              APPLICATION NUMBER: 07/950,417
              FILING DATE: 23-Sep-1992
         ATTORNEY/AGENT INFORMATION:
              NAME: Dwyer, Patrick M.
              REGISTRATION NUMBER: 32,411
              REFERENCE/DOCKET NUMBER: PROTEO.POOC1
         TELECOMMUNICATION INFORMATION:
              TELEPHONE: (206) 343-7074
              TELEFAX: (206) 343-7085
    INFORMATION FOR SEQ ID NO: 2:
         SEQUENCE CHARACTERISTICS:
              LENGTH: 28 AMINO ACIDS
```

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TYPE: AMINO ACID
              STRANDEDNESS: SINGLE
              TOPOLOGY: LINEAR
        MOLECULE TYPE: PEPTIDE
              DESCRIPTION: /A4 (1-28); page 83, line 31
         SEQUENCE DESCRIPTION: SEQ ID NO: 2:
US-08-723-661B-2
                          85.4%; Score 35; DB 4; Length 28;
 Query Match
 Best Local Similarity
                          100.0%; Pred. No. 1.3;
 Matches
             7; Conservative
                                 0; Mismatches
                                                    0;
                                                                              0;
                                                        Indels
                                                                  0;
                                                                      Gaps
            1 LVFFAED 7
Qу
              111111
           17 LVFFAED 23
Db
RESULT 46
US-09-660-954-2
; Sequence 2, Application US/09660954
; Patent No. 6471960
   GENERAL INFORMATION:
         APPLICANT: ANDERSON, STEPHEN
         TITLE OF INVENTION: METHODS FOR THE PREVENTION AND TREATMENT
                             OF VASCULAR HEMORRHAGING AND ALZHEIMER'S DISEASE
         NUMBER OF SEQUENCES: 14
         CORRESPONDENCE ADDRESS:
              ADDRESSEE: HOWREY & SIMON
              STREET: 1299 PENNSYLVANIA AVENUE, N.W.
              CITY: WASHINGTON
              STATE: D.C.
              COUNTRY: US
              ZIP: 20004
         COMPUTER READABLE FORM:
              MEDIUM TYPE: Floppy disk
              COMPUTER: IBM PC compatible
              OPERATING SYSTEM: PC-DOS/MS-DOS
              SOFTWARE: PatentIn Release #1.0, Version #1.25
         CURRENT APPLICATION DATA:
              APPLICATION NUMBER: US/09/660,954
              FILING DATE: 13-Sep-2000
              CLASSIFICATION: <Unknown>
         PRIOR APPLICATION DATA:
              APPLICATION NUMBER: US/09/388,890
              FILING DATE: <Unknown>
              APPLICATION NUMBER: 08/686,959
              FILING DATE: <Unknown>
         ATTORNEY/AGENT INFORMATION:
              NAME: AUERBACH, JEFFREY I.
              REGISTRATION NUMBER: 32,680
         TELECOMMUNICATION INFORMATION:
              TELEPHONE: (202) 383-7451
              TELEFAX: (202) 383-6610
    INFORMATION FOR SEQ ID NO: 2:
         SEQUENCE CHARACTERISTICS:
;
              LENGTH: 28 amino acids
              TYPE: amino acid
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TOPOLOGY: linear
         MOLECULE TYPE: peptide
         HYPOTHETICAL: NO
         FRAGMENT TYPE: N-terminal
         ORIGINAL SOURCE:
              ORGANISM: HOMO SAPIENS
         IMMEDIATE SOURCE:
              CLONE: B(1-28) peptide of amyloid B protein
         SEQUENCE DESCRIPTION: SEQ ID NO: 2:
US-09-660-954-2
                          85.4%; Score 35; DB 4; Length 28; 100.0%; Pred. No. 1.3;
  Query Match
  Best Local Similarity
            7; Conservative 0; Mismatches
                                                    0; Indels
                                                                  0; Gaps
                                                                               0;
            1 LVFFAED 7
Qу
              111111
           17 LVFFAED 23
RESULT 47
US-09-660-954-3
; Sequence 3, Application US/09660954
 Patent No. 6471960
    GENERAL INFORMATION:
         APPLICANT: ANDERSON, STEPHEN
         TITLE OF INVENTION: METHODS FOR THE PREVENTION AND TREATMENT
                             OF VASCULAR HEMORRHAGING AND ALZHEIMER'S DISEASE
         NUMBER OF SEQUENCES: 14
         CORRESPONDENCE ADDRESS:
              ADDRESSEE: HOWREY & SIMON
              STREET: 1299 PENNSYLVANIA AVENUE, N.W.
              CITY: WASHINGTON
              STATE: D.C.
              COUNTRY: US
              ZIP: 20004
         COMPUTER READABLE FORM:
              MEDIUM TYPE: Floppy disk
              COMPUTER: IBM PC compatible
             OPERATING SYSTEM: PC-DOS/MS-DOS
              SOFTWARE: PatentIn Release #1.0, Version #1.25
         CURRENT APPLICATION DATA:
              APPLICATION NUMBER: US/09/660,954
              FILING DATE: 13-Sep-2000
              CLASSIFICATION: <Unknown>
         PRIOR APPLICATION DATA:
              APPLICATION NUMBER: US/09/388,890
              FILING DATE: <Unknown>
              APPLICATION NUMBER: 08/686,959
              FILING DATE: <Unknown>
         ATTORNEY/AGENT INFORMATION:
              NAME: AUERBACH, JEFFREY I.
              REGISTRATION NUMBER: 32,680
         TELECOMMUNICATION INFORMATION:
              TELEPHONE: (202) 383-7451
              TELEFAX: (202) 383-6610
    INFORMATION FOR SEQ ID NO: 3:
```

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SEQUENCE CHARACTERISTICS:
              LENGTH: 28 amino acids
              TYPE: amino acid
              TOPOLOGY: linear
         MOLECULE TYPE: peptide
         HYPOTHETICAL: YES
         FRAGMENT TYPE: N-terminal
         ORIGINAL SOURCE:
              ORGANISM: HOMO SAPIENS
         IMMEDIATE SOURCE:
              CLONE: DlN B(1-28) peptide of amyloid B protein
         SEQUENCE DESCRIPTION: SEQ ID NO: 3:
US-09-660-954-3
  Query Match
                          85.4%; Score 35; DB 4; Length 28;
  Best Local Similarity 100.0%; Pred. No. 1.3;
  Matches 7; Conservative 0; Mismatches
                                                   0; Indels
                                                                 0; Gaps
                                                                              0;
           1 LVFFAED 7
Qу
              111111
           17 LVFFAED 23
Db
RESULT 48
US-09-660-954-4
; Sequence 4, Application US/09660954
; Patent No. 6471960
    GENERAL INFORMATION:
         APPLICANT: ANDERSON, STEPHEN
         TITLE OF INVENTION: METHODS FOR THE PREVENTION AND TREATMENT
                             OF VASCULAR HEMORRHAGING AND ALZHEIMER'S DISEASE
         NUMBER OF SEQUENCES: 14
         CORRESPONDENCE ADDRESS:
              ADDRESSEE: HOWREY & SIMON
              STREET: 1299 PENNSYLVANIA AVENUE, N.W.
              CITY: WASHINGTON
              STATE: D.C.
              COUNTRY: US
              ZIP: 20004
         COMPUTER READABLE FORM:
              MEDIUM TYPE: Floppy disk
              COMPUTER: IBM PC compatible
              OPERATING SYSTEM: PC-DOS/MS-DOS
              SOFTWARE: PatentIn Release #1.0, Version #1.25
         CURRENT APPLICATION DATA:
              APPLICATION NUMBER: US/09/660,954
              FILING DATE: 13-Sep-2000
              CLASSIFICATION: <Unknown>
         PRIOR APPLICATION DATA:
              APPLICATION NUMBER: US/09/388,890
              FILING DATE: <Unknown>
              APPLICATION NUMBER: 08/686,959
              FILING DATE: <Unknown>
         ATTORNEY/AGENT INFORMATION:
              NAME: AUERBACH, JEFFREY I.
              REGISTRATION NUMBER: 32,680
         TELECOMMUNICATION INFORMATION:
```

```
TELEPHONE: (202) 383-7451
              TELEFAX: (202) 383-6610
    INFORMATION FOR SEQ ID NO: 4:
         SEQUENCE CHARACTERISTICS:
              LENGTH: 28 amino acids
              TYPE: amino acid
              TOPOLOGY: linear
         MOLECULE TYPE: peptide
         HYPOTHETICAL: YES
         FRAGMENT TYPE: N-terminal
         ORIGINAL SOURCE:
              ORGANISM: HOMO SAPIENS
         IMMEDIATE SOURCE:
              CLONE: E3Q B(1-28) peptide of amyloid B protein
         SEQUENCE DESCRIPTION: SEQ ID NO: 4:
US-09-660-954-4
  Query Match
                          85.4%; Score 35; DB 4; Length 28;
  Best Local Similarity
                          100.0%; Pred. No. 1.3;
 Matches
            7; Conservative
                                0; Mismatches
                                                   0; Indels
                                                                  0; Gaps
                                                                              0;
Qy
            1 LVFFAED 7
              1111111
           17 LVFFAED 23
RESULT 49
US-09-660-954-5
; Sequence 5, Application US/09660954
 Patent No. 6471960
    GENERAL INFORMATION:
         APPLICANT: ANDERSON, STEPHEN
         TITLE OF INVENTION: METHODS FOR THE PREVENTION AND TREATMENT
                             OF VASCULAR HEMORRHAGING AND ALZHEIMER'S DISEASE
         NUMBER OF SEQUENCES: 14
         CORRESPONDENCE ADDRESS:
              ADDRESSEE: HOWREY & SIMON
              STREET: 1299 PENNSYLVANIA AVENUE, N.W.
              CITY: WASHINGTON
              STATE: D.C.
              COUNTRY: US
              ZIP: 20004
         COMPUTER READABLE FORM:
              MEDIUM TYPE: Floppy disk
              COMPUTER: IBM PC compatible
              OPERATING SYSTEM: PC-DOS/MS-DOS
              SOFTWARE: PatentIn Release #1.0, Version #1.25
         CURRENT APPLICATION DATA:
              APPLICATION NUMBER: US/09/660,954
              FILING DATE: 13-Sep-2000
              CLASSIFICATION: <Unknown>
         PRIOR APPLICATION DATA:
              APPLICATION NUMBER: US/09/388,890
              FILING DATE: <Unknown>
              APPLICATION NUMBER: 08/686,959
              FILING DATE: <Unknown>
         ATTORNEY/AGENT INFORMATION:
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NAME: AUERBACH, JEFFREY I.
              REGISTRATION NUMBER: 32,680
         TELECOMMUNICATION INFORMATION:
              TELEPHONE: (202) 383-7451
              TELEFAX: (202) 383-6610
    INFORMATION FOR SEQ ID NO: 5:
         SEQUENCE CHARACTERISTICS:
              LENGTH: 28 amino acids
              TYPE: amino acid
              TOPOLOGY: linear
         MOLECULE TYPE: peptide
         HYPOTHETICAL: YES
         FRAGMENT TYPE: N-terminal
         ORIGINAL SOURCE:
              ORGANISM: HOMO SAPIENS
         IMMEDIATE SOURCE:
              CLONE: R5Q B(1-28) peptide of amyloid B protein
         SEQUENCE DESCRIPTION: SEQ ID NO: 5:
US-09-660-954-5
 Query Match 85.4%; Score 35; DB 4; Length 28; Best Local Similarity 100.0%; Pred. No. 1.3;
          7; Conservative 0; Mismatches 0; Indels
                                                                  0; Gaps
                                                                               0;
Qу
            1 LVFFAED 7
              Db
           17 LVFFAED 23
RESULT 50
US-09-660-954-6
; Sequence 6, Application US/09660954
; Patent No. 6471960
    GENERAL INFORMATION:
         APPLICANT: ANDERSON, STEPHEN
         TITLE OF INVENTION: METHODS FOR THE PREVENTION AND TREATMENT
                             OF VASCULAR HEMORRHAGING AND ALZHEIMER'S DISEASE
         NUMBER OF SEQUENCES: 14
         CORRESPONDENCE ADDRESS:
              ADDRESSEE: HOWREY & SIMON
              STREET: 1299 PENNSYLVANIA AVENUE, N.W.
              CITY: WASHINGTON
              STATE: D.C.
              COUNTRY: US
              ZIP: 20004
         COMPUTER READABLE FORM:
              MEDIUM TYPE: Floppy disk
              COMPUTER: IBM PC compatible
              OPERATING SYSTEM: PC-DOS/MS-DOS
              SOFTWARE: PatentIn Release #1.0, Version #1.25
         CURRENT APPLICATION DATA:
              APPLICATION NUMBER: US/09/660,954
              FILING DATE: 13-Sep-2000
              CLASSIFICATION: <Unknown>
         PRIOR APPLICATION DATA:
              APPLICATION NUMBER: US/09/388,890
              FILING DATE: <Unknown>
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APPLICATION NUMBER: 08/686,959
              FILING DATE: <Unknown>
         ATTORNEY/AGENT INFORMATION:
              NAME: AUERBACH, JEFFREY I.
              REGISTRATION NUMBER: 32,680
         TELECOMMUNICATION INFORMATION:
              TELEPHONE: (202) 383-7451
              TELEFAX: (202) 383-6610
    INFORMATION FOR SEQ ID NO: 6:
         SEQUENCE CHARACTERISTICS:
              LENGTH: 28 amino acids
              TYPE: amino acid
              TOPOLOGY: linear
         MOLECULE TYPE: peptide
         HYPOTHETICAL: YES
         FRAGMENT TYPE: N-terminal
         ORIGINAL SOURCE:
              ORGANISM: HOMO SAPIENS
         IMMEDIATE SOURCE:
              CLONE: H6Q B(1-28) peptide of amyloid B protein
         SEQUENCE DESCRIPTION: SEQ ID NO: 6:
US-09-660-954-6
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                          85.4%; Score 35; DB 4; Length 28;
  Best Local Similarity 100.0%; Pred. No. 1.3;
  Matches
          7; Conservative
                              0; Mismatches
                                                   0; Indels
                                                                 0; Gaps
                                                                              0;
           1 LVFFAED 7
Qγ
              111111
Db
           17 LVFFAED 23
RESULT 51
US-09-660-954-7
; Sequence 7, Application US/09660954
; Patent No. 6471960
    GENERAL INFORMATION:
         APPLICANT: ANDERSON, STEPHEN
         TITLE OF INVENTION: METHODS FOR THE PREVENTION AND TREATMENT
                             OF VASCULAR HEMORRHAGING AND ALZHEIMER'S DISEASE
         NUMBER OF SEQUENCES: 14
         CORRESPONDENCE ADDRESS:
              ADDRESSEE: HOWREY & SIMON
              STREET: 1299 PENNSYLVANIA AVENUE, N.W.
              CITY: WASHINGTON
              STATE: D.C.
              COUNTRY: US
              ZIP: 20004
         COMPUTER READABLE FORM:
              MEDIUM TYPE: Floppy disk
              COMPUTER: IBM PC compatible
              OPERATING SYSTEM: PC-DOS/MS-DOS
              SOFTWARE: PatentIn Release #1.0, Version #1.25
         CURRENT APPLICATION DATA:
              APPLICATION NUMBER: US/09/660,954
              FILING DATE: 13-Sep-2000
              CLASSIFICATION: <Unknown>
```

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PRIOR APPLICATION DATA:
             APPLICATION NUMBER: US/09/388,890
              FILING DATE: <Unknown>
             APPLICATION NUMBER: 08/686,959
              FILING DATE: <Unknown>
         ATTORNEY/AGENT INFORMATION:
              NAME: AUERBACH, JEFFREY I.
              REGISTRATION NUMBER: 32,680
         TELECOMMUNICATION INFORMATION:
              TELEPHONE: (202) 383-7451
              TELEFAX: (202) 383-6610
    INFORMATION FOR SEQ ID NO: 7:
         SEQUENCE CHARACTERISTICS:
              LENGTH: 28 amino acids
              TYPE: amino acid
;
             TOPOLOGY: linear
        MOLECULE TYPE: peptide
        HYPOTHETICAL: YES
         FRAGMENT TYPE: N-terminal
        ORIGINAL SOURCE:
             ORGANISM: HOMO SAPIENS
         IMMEDIATE SOURCE:
              CLONE: D7Q B(1-28) peptide of amyloid B protein
         SEQUENCE DESCRIPTION: SEQ ID NO: 7:
US-09-660-954-7
 Query Match
                          85.4%; Score 35; DB 4; Length 28;
 Best Local Similarity 100.0%; Pred. No. 1.3;
 Matches
                               0; Mismatches
            7; Conservative
                                                   0; Indels
                                                                 0; Gaps
                                                                             0;
           1 LVFFAED 7
Qу
              Db
          17 LVFFAED 23
RESULT 52
US-09-660-954-8
; Sequence 8, Application US/09660954
 Patent No. 6471960
   GENERAL INFORMATION:
        APPLICANT: ANDERSON, STEPHEN
        TITLE OF INVENTION: METHODS FOR THE PREVENTION AND TREATMENT
                            OF VASCULAR HEMORRHAGING AND ALZHEIMER'S DISEASE
        NUMBER OF SEQUENCES: 14
        CORRESPONDENCE ADDRESS:
             ADDRESSEE: HOWREY & SIMON
             STREET: 1299 PENNSYLVANIA AVENUE, N.W.
             CITY: WASHINGTON
             STATE: D.C.
             COUNTRY: US
             ZIP: 20004
        COMPUTER READABLE FORM:
             MEDIUM TYPE: Floppy disk
             COMPUTER: IBM PC compatible
             OPERATING SYSTEM: PC-DOS/MS-DOS
             SOFTWARE: PatentIn Release #1.0, Version #1.25
        CURRENT APPLICATION DATA:
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